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LXXXI.

FACTS OF AUDITION.

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It is true that the physiology of the cochlea still remains a bewildering subject. However, many things that were bewildering ten years ago are now readily explained and comprehensible. Since the days of Helmholtz and his contemporary workers little has been added to the knowledge of auditory function until recently. In the past few years much work has been done by physicists, physiologists and psychologists, particularly in the application of audion bulb circuits to the auditory problem. This work has thrown new light on this subject.

The subject of the physiology of the cochlea might conveniently be subdivided into two headings: the facts of audition and the theories of audition. We are sure of one thing: whatever is known or ever will be known regarding cochlear function must be based upon observed facts. Every theory of the function of hearing is the outgrowth of an attempt to explain these facts. Several plausible theories have already been advanced. These are capable of explaining many of the observed

facts. For example, if we employ such simple analogies as the harp and the telephone, many of the facts of audition become explicable. Up to the present time, however, there is no theory of audition that has received general acceptance. Ultimately a sufficient number of facts may lead to an entirely satisfactory theory. In the meantime, in actual otologic practice it is helpful for us to utilize what is already available. A knowledge of the already established facts, and a familiarity with at least the most plausible theories, are helpful in attacking auditory problems which are met in daily practice. For example, in order to determine the status of a hearing defect, one needs to understand the physical properties of loudness and pitch. Thus, the degree of any hearing defect is measured by the degree of loudness with which tones are heard. In a similar way, the determination of the type of any hearing defect is facilitated by obtaining the lower and upper limits of pitch.

The present communication will be limited to a presentation of certain salient facts which have a practical value in clinical practice. It is planned in a subsequent communication to discuss the theories of audition.

FACTS OF AUDITION.

Although, to be sure, many of the facts of audition are as old or older than Helmholtz, yet there are many new facts of practical value. It is also true that even the old facts have a new value in the light of recent developments. In an attempt to understand auditory function, the most concrete information may be obtained by regarding the ear as a physical instrument. In determining the characteristics of any physical instrument in the laboratory, quantitative data are obtained, which give the relation of the response of the instrument to definite stimuli. The value of the stimulus necessary just barely to elicit a response must be known. This serves as a measure of the "sensitiveness" of the instrument. Also the minimal differences detectable by the instrument must be determined. This gives a measure of the "sensibility." Finally, it is necessary to determine the upper limit of the intensity of stimuli the instrument will accommodate. To illustrate: to determine the characteristics of a sensitive galvanometer, one determines first how small an electric current it will detect; second, how

small a difference in the strength of the current it will detect, and third, how large a current it will accommodate. The minuteness of the current it will detect is a measure of its "sensitiveness." The minuteness of the differences of current it can detect is a measure of its "sensibility." The minimal and maximal currents that it will accommodate are the measure of its range of usefulness.

Employing this method of approach, we may perhaps gain a clearer conception of the physiology of the cochlea. Measurements upon the ear as a physical instrument have brought out certain facts. A mere outline of certain of these facts will serve the present purpose.

The Physical Properties of Sound.—Whenever a wave-motion of a certain intensity and frequency is conducted to the cochlea, there results a sensation of sound. If this wave-motion consists of a regular succession of vibrations, the sensation is that of a tone. Whenever this wave-motion consists of an irregular succession of vibrations there results a sensation of noise. A tone, therefore, is a result of a periodic wave-motion. A noise is a result of a nonperiodic wave-motion. A sound, therefore, may be classified either as a tone or a noise.

Consideration should be given to two aspects of this appreciation of sound. First, the physical characteristics of the wave-motion in the external world; and second, the response of the cochlea to these wave-motions. The external wave-motion producing the sound has three distinct properties. These are amplitude, frequency and wave-form. The amplitude of the wave-motion determines the loudness of the sound sensation. The frequency of the wave-motion determines the pitch of the sound sensation. The wave-form determines the quality of the sound sensation. As the amplitude of the wave-motion increases, the loudness of the sensation increases. As the frequency of the wave-motion increases, the pitch of the sensation rises. Thus, the amplitude determines the loudness and the frequency determines the pitch.

The relation of the response of the cochlea to the external stimulus, however, is not one of a simple direct proportion. For example, the increase in the amplitude of from 10 to 100 produces the same increase in loudness sensation as the increase in the amplitude of from 100 to 1000. This relation

between the stimulus and the cochlear response is characteristic of other sense organs and is known as the Weber-Fechner law of psychophysics.

The relation of the frequency of the wave-motion to the pitch sensation is well known. There is the same interval of pitch between 32 and 64 d. v. that there is between 4096 and 8192 d. v. This interval of pitch is the octave. This musical interval of pitch, the octave, is produced by two tones whose frequencies have the ratio of one to two. Equal ratios of frequencies produce equal intervals of pitch.

LOUDNESS.

Before a tone can be sensed, the vibrations must attain a certain amplitude. That is, the energy of the wave-motion reaching the cochlea must attain a definite minimal level. The requisite amplitude or energy varies greatly for tones of different pitch. This is true of the normal or the pathologic cochlea. Tones of low pitch require relatively great energy. Tones of moderately high pitch require a much smaller energy. Tones of highest pitch, however, again require a greater energy. For example, in order to produce a barely audible tone of 32 d. v., the energy must be one million times that required to produce a barely audible tone of 1024 d. v. In order to produce a barely audible tone of 16,384 d. v., the energy must be ten thousand times that required to produce the barely audible tone of 1024 d. v. It would seem that we have this fortunate adaptation, whereby the cochlea is most easily affected by tones of the frequency range employed in speech. In any given individual, the amount of energy in the wave-motion, and the acuity of the hearing of the individual, determine the loudness of the sensation of tone. One of the best methods of testing auditory acuity is the measurement of this minimal threshold of loudness. Such measurements of loudness are basic in determining the nature and degree of a hearing defect.

As the amplitude is increased, the tone becomes louder and louder, until it attains the upper limit of loudness. This upper limit of loudness is reached when a greater increase of amplitude does not increase the loudness sensation. At this loudness level the tone becomes "painfully loud." If carried beyond this limit, there is not only the sensation of pain but also the tactile

sensation of feeling. For example, even a deafmute, though he has no sensation of tone, is able to feel such excessive stimulation.

The Sensibility of the Cochlea in Discriminating Differences of Loudness.—Between the lower and upper thresholds of loudness, the human cochlea can appreciate many gradations of loudness. For example, the normal human ear can appreciate about 65 gradations of loudness for a tone of 32 d. v. For a tone of 512 d. v., however, approximately 270 gradations of loudness can be appreciated. For very high tones, again, a fewer number of gradations of loudness can be appreciated. For very low tones and for very high tones the lower limit of loudness and the upper limit of loudness are relatively near. For tones in the middle register of pitch, the lower limit and the upper limit of loudness are relatively far apart.

In order that one tone can be appreciated as louder than another, the amplitude must be increased at least 5 per cent. An impaired cochlea can appreciate increments of loudness just as well as the normal cochlea. A lesion of either the middle or the internal ear has no effect upon the capability of the cochlea to differentiate loudness.

The sensitivity of the ear to loudness is represented graphically in Fig. 1. This chart shows the lower and upper limits of loudness for the average normal.

PITCH.

The range of pitch to which the human ear responds is from 20 to 20,000 d. v. This embraces a range of ten octaves. This range appears quite remarkable when we consider that the eye responds to a single octave. The lowest frequency to which the eye responds is about four hundred million vibrations per second. This produces a color sensation of red. The highest frequency to which the eye responds is about eight hundred million vibrations per second. This produces a color sensation of violet. The visible range is, therefore, only a single octave.

In the ten octaves which the ear appreciates, the normal cochlea can distinguish about 1500 gradations of pitch. The ear can just barely distinguish between tones of 128 and 129 d. v. It can just barely distinguish between tones of 1000 and

1003 d. v. It can just barely distinguish between tones of 4000 and 4013 d. v.

Pathologic changes do not affect the ability of the cochlea to sense differences of pitch; the pathologic cochlea can perceive differences of pitch as well as can the normal cochlea. A person who is musically trained, or has musical talent, cannot sense any smaller differences of pitch than can the untrained or untalented. He can, however, detect with greater nicety whether any given tone is sharpened or flattened.

The lower and upper limits of pitch are also represented graphically in Fig. 1. This figure shows the entire gamut of tones to which the ear responds. The area between the two curves shows the range of loudness and the range of pitch to which the normal ear responds. This area is spoken of as the "auditory sensation area." The lower and upper limits of pitch are indicated at the points of intersection of these two curves. If the amplitude and frequency of a wave-motion is such as to come within the limits of these two curves, it will be sensed by the average normal ear as a tone. If it falls outside the boundary of these two curves, it will not be sensed as a tone.

If we consider the total number of gradations of loudness and the total number of gradations of pitch to which the normal ear responds, we attain the number of pure tones the normal ear can appreciate. This number is approximately 324,000. The number of these pure tones which can be sensed by any given individual is a measure of his percentage of hearing. Thus, if a person with impaired hearing can appreciate 162,000 pure tones, he possesses, according to this scale, 50 per cent of normal.

QUALITY.

The third property of tone is quality. The quality of a musical tone is determined by the shape of the wave-form. The wave-forms of sounds produced by a tuning fork, a violin and a flute are represented in Fig. 2.

The tone produced by a tuning fork is a near approach to a pure tone. As will be noticed, its form is the simplest of the three. Such a simple pendular wave-form is the distinguishing characteristic of a pure tone. It will be noted that

the wave-form of the tones produced by the violin and the flute are more complex. The loudness and pitch of these three tones are the same. They have the same amplitude and they have the same frequency. It is the difference of wave-form that produces the difference in quality. Expressed in another but equivalent manner, the quality of a musical tone is determined by the number and relative loudness of the overtones which accompany it. If it has no overtones, it is a pure tone. In general, overtones consist of tones whose frequencies are 2, 3, 4, 5, 6, etc., times the frequency of the fundamental tone. For example, in the case of the violin tone represented in Fig. 2, the fundamental tone has a frequency of 256 d. v. The first overtone has a frequency of two times 256, i. e., 512 d. v. The second overtone has a frequency of three times 256, i. e., 768 d. v. The third overtone has a frequency of four times 256, i. e., 1024 d. v.

For some complex tones, it is possible to identify as many as forty such overtones. It is the number and prominence of such overtones that determine the quality of a musical tone. This may be demonstrated by referring to two simple experiments. One listens to a violin tone received by a radio set. In the receiving circuit one introduces an electric filter, which will let pass any desired frequencies contained in this violin tone. By filtering out all overtones one obtains a pure tone that sounds exactly like a tuning fork. By filtering certain of the overtones and not others, one can obtain tones of distinctly varied quality. This demonstrates that quality depends upon the number and prominence of overtones. Again, in addition to this analytic method, the same cardinal principle of quality can be demonstrated synthetically. By sounding different combinations of harmonic tuning forks, one can build up tonal qualities characteristic of many musical instruments, such as the violin, the cornet, the flute, the oboe and the clarinet.

MISCELLANEOUS FACTS.

Certain other facts of audition should be mentioned in outline.

Masking of Tones.—It is a commonly observed fact that the presence of one tone will interfere with the hearing of another tone. For example, two tuning forks of different frequencies

are sounded together. One hears both tones, provided they are sufficiently loud. As they diminish in loudness, however, there will come a time when the tone of higher pitch is no longer heard. If, however, the fork producing the lower tone be stopped, one is surprised at the loudness of the tone being produced by the higher fork. The tone of higher pitch was not heard in the presence of the tone of lower pitch, but as soon as the lower tone ceased, the higher tone was quite prominent. This obliterating of one tone by another is known as "auditory masking." Such masking is useful in tests for malingerers. It is also one of the useful facts in formulating a plausible theory of hearing.

Beats.—Two tones differing slightly in pitch, sounded together, produce "beats." Thus, two tones of 256 and 257 d. v., if sounded together, unite so as periodically to augment and diminish in loudness. This augmentation and diminution in loudness will occur once each second, since they differ by one vibration per second. If the two tones are 256 and 266 d. v., the two tones unite so as to augment and diminish in loudness ten times per second. This produces ten beats per second. If these beats are more frequent than twenty per second they themselves will be sensed as a musical tone. In other words, the frequency of these beats has reached the pitch threshold of audibility, and therefore is sensed as a tone. It is to be noted that such beats produce another low tone in addition to the tones produced by the tuning fork. Each tuning fork produces its own tone. Sounded together, they produce beats, which produce another lower tone.

Subjective Tones.—If two tones, say of 200 and 300 d. v., are heard together, the cochlea will sense not only these two tones of 200 and 300 d. v., but in addition will sense tones of 500 d. v. and 100 d. v. These tones of 500 and 100 d. v. are called subjective tones. They are, respectively, the summation and the difference of the original two tones of 200 d. v. and 300 d. v. These tones are called "subjective" tones because they have no counterpart in the external world. Actually they are not necessarily subjective in the sense of being cerebral. Experiments in physics have shown that the production of these tones can be explained by the action of the middle and

internal ear mechanisms and that it is not necessary to postulate a cortical explanation.

Localization of Sound.—Although it is possible for a single ear to give some information regarding the direction and distance of the source of a sound, much greater accuracy is attained by the use of both ears. When one is hearing with both ears he attains, as it were, an "auditory perspective." The impressions received by one ear may be slightly different from the impressions received by the other. There may be a difference in loudness, or in direction, or in "phase" (time-relation). Because of one or more of these factors, two ears are enabled to locate sound better than one ear. In fact, an individual with hearing in only one ear is very inaccurate in his localization of sound. This fact can be made use of in tests for malingering.

The Nature of Vowels and Consonants.—Ordinary conversational speech consists of modulated tones and noises. In the production of speech, tones or noises are produced by the larynx. These tones or noises are modulated and altered by cavities of the nose, throat and mouth, and the stopping effects of the teeth, lips and tongue. Vowel sounds consist essentially of sustained vibrations. These vibrations first build up to a certain value, then are sustained for a short interval, and then diminish to inaudibility. The frequencies of vowel sounds embrace a range between about 200 and 2000 d. v. Consonants, on the other hand, are not sustained to any great length and consist essentially of high frequency vibrations. For example, the "s" sound, the "th" sound and the "f" sound are made up essentially of frequencies above 2000 d. v. Some consonant sounds may require frequencies even higher than 5000 d. v. For the perfect appreciation of speech, a person should have approximately normal hearing for limits of pitch between 60 and 6000 d. v.

Music, on the other hand, requires a greater range of pitch appreciation, both above and below the range required for speech. For the full appreciation of music, the ear should respond to a tonal range between the limits of about 30 to 16,000 d. v. In this respect an individual with impaired hearing may be at a greater disadvantage for appreciation of music than for conversational speech.

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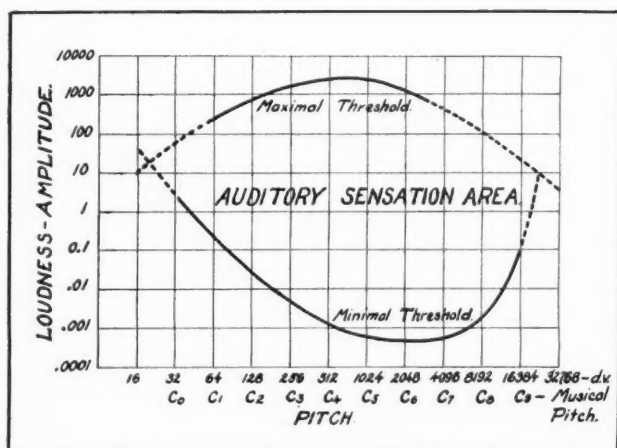


Fig. 1.

Curves showing the minimal and maximal thresholds of loudness for tones throughout the entire audible pitch range for the average normally hearing individual (adapted from the curves of Fletcher and Wegel).

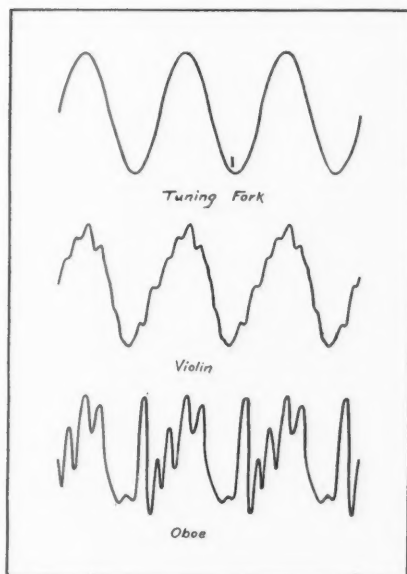


Fig. 2

Curves illustrating the differences in quality of tones of the same pitch and loudness, produced by a tuning fork, a violin and an oboe.

LXXXII.

CYSTS IN THE FLOOR OF THE MOUTH.*

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Rarely are cysts involving the floor of the mouth a subject for report or discussion by laryngologists, but usually the cases that have been seen and studied are those of the general and the oral surgeons. Nevertheless, cysts in this region are not infrequently seen by the laryngologist, and, because of this, as well as their general interest, the subject may warrant a presentation.

Considering the present advanced state of medicine, it surprises the reflective mind how little is known, by many, of the true character of these tumors, and also how meagerly the subject is treated, if at all, by most of the textbooks and books of reference.

There is quite a variety of these cysts, including ranula, dermoid, thyroglossal, branchial, multilocular, mucous and echinococcus, but a satisfactory classification for a basis of study has not been found. This has been due partly to the manner in which cases have been reported, and partly to the incomplete study given to the origin of the growth. A more comprehensive understanding of their true character might best be obtained if greater consideration were given to the kind of tissue from which they originate, with a minute examination of their saclike walls. In other words, a gross anatomic and histologic or histopathologic study must be made instead of a report on the cyst contents alone. In determining the parentage of such cysts, not only must the anatomic structures composing the floor of the mouth be considered, but the neighboring structures that occasionally invade or encroach upon this territory must be thought of; such as the origin of the branchiogenetic, the thyroglossal and the jawbone cysts.

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The floor of the mouth is not a large territory, but owing to the movability of the tongue and the elasticity of the soft tissues it is often remarkable what size such cysts may attain, occasionally approaching that of a small fist or even larger. The tongue forced against the roof of the mouth may limit the upward growth, but they may present well below the chin and even into the neck. Usually they are much smaller than this—from walnut to hen's egg size, while some of the mucous cysts may not be larger than a filbert or pea. In general they are smooth, globular, glistening and centrally or laterally located, easily movable or displaced, if not too large, tense to palpation, as a rule painless, and inconvenience chiefly from their size, and are not accompanied by neighboring adenitis. Their growth is slow, and they occur in male and female alike and at all ages.

The most important cysts will be discussed in detail and in the order of their frequency.

Ranula.—Although mentioned by our very earliest confreres, ranula was little understood until recent years. Many of the cysts found in this region were generally called ranula, and though some considerable study had been given to their origin, a diversity of opinion existed that far from clarified the situation. It was customary to believe, and the general understanding seemed to be, that a ranula was any cyst in the floor of the mouth containing mucus or saliva, the result of a retention caused by the closure of the duct of a mucous or salivary gland. The failure of the secretion to escape from the orifice of the duct was attributed either to an inflammation of its lining membrane, with subsequent swelling and obstruction, or to an increase in the viscosity of the fluid. In some instances precipitation of the salts and the formation of a calculus was thought to bring about the closure. Anything that might cause a stricture of the duct or a plugging of its outlet, like a cicatrix resulting from an incision or injury, or an ulcer, or pressure from a tumor or mass upon the duct and occluding it, was assumed to result in retention of its secreting fluid with consequent dilatation and cyst formation.

Diemerbroeck¹ was the earliest observer who attempted to show that ranula resulted from the retention of saliva in the duct of the submaxillary gland. Much later, Dupuytren² ex-

plained their presence as a result of mucus retained in the mucous glands of the mouth. Fleischmann³ confined their origin to an obstruction to the flow of mucus from a certain follicle that he discovered on the floor of the mouth, while Neumann⁴ thought the gland tubules of Bochdoleck were the seat of origin. Pauli⁵ described a most interesting observation wherein a stenosis of Wharton's duct produced a dilatation of the same until the thin wall ruptured, allowing the retained saliva to extravasate into the neighboring connective tissue, creating a *ptylectasia*, and, several of these uniting, formed a *ptylocele*. There is a cluster of mucous glands beneath the tip of the tongue called the Blandin-Nuhn gland. Von Recklinghausen⁶ expressed his belief that obstruction to the duct of this gland resulted in a retention mucous cyst known as *ranula*.

The sublingual salivary gland has many small ducts called Rivini, besides the main large one called the duct of Bartholin. The smaller ones open on a ridge alongside of the frenum of the tongue, while the duct of Bartholin opens into the floor of the mouth near the frenum, either separately as a small papilla, or as is more usual, in common with the duct from the submaxillary salivary gland, known as the duct of Wharton. Obstruction in any of these ducts resulting in retention of saliva was thought by others to be the only cause of this condition. As late as 1904 Deaver⁷ states that an obstruction and dilatation of one of the several ducts opening at the side of the lingual frenum will occasion a cystic swelling known as *ranula*; also, it may be due to an obstructed mucous follicle.

Suzanne⁸ and Hippel⁹ appear to be the first to recognize the sublingual gland as the site of this cyst. Hippel more than anyone else championed this view, and his studies of the histopathology brought conclusive evidence as proof of his position. The experimental work of De Closmadene, Cl. Bernard, Cornil, Ranvier and Suzanne demonstrated that no cyst resulted following ligation or cautery of any of these ducts. On the contrary, atrophy and cessation of all gland secretion resulted, with no *ranula*. Hippel's investigation then demonstrated a chronic interstitial inflammation of the gland with subsequent closure of the ducts. The lining epithelium in these ducts proliferate, then desquamate. The accumulated salivary secretion,

plus the transudated capillary secretion, produces a pressure atrophy of the gland tissue, causing it to disappear, eventually being replaced by connective tissue. Hippel's findings are based on the histopathologic study of seven cases. All of these showed the inner wall of each cyst made up of two different layers of cells, mostly spindle form, and in between large round cells and a few small round cells. Besides these were other large round protoplasmic cells, arranged in irregular manner. There was neither basement membrane nor epithelial cells of any description present.

Thompson¹⁰ recognizes a cyst of the submaxillary gland and a cyst of the sublingual, but claims a direct relationship exists between these and a branchiogenetic cyst. In other words, they are compound cysts arising from a persistent cervical sinus and arriving in the submaxillary area by migration, the result of muscle action.

Although the term ranula is applied by many to a large variety of cysts, including the parotid and even the pancreatic duct, its definition should be confined to a degenerative cyst formation of the salivary glands in the sublingual region. Its histopathology shows it to be the result of a degenerative process of a salivary gland. Its chronicity, the absence of a mechanical obstruction of its orifices, its tendency to recur and its usual position to one side of the frenum help to differentiate it.

Hygromas that result from the occlusion of the ducts or orifices of mucous glands found in this region of the mouth—as the glandula incisiva, the gland of Blandin-Nuhn, beneath the tip of the tongue, Fleischmann's bursa, etc.—have a different pathology. These cysts are in the mucous membrane, while ranulae are beneath the mucous membrane. The wall of the mucous cyst is lined with epithelium and contains lymph structures. They are true retention cysts, usually the size of a pea or bean, and occur single or multiple in any part of the mucous membrane of the mouth.

A condition described as acute ranula is occasionally seen. It occurs rather suddenly, probably while masticating. There is sudden pain and the appearance of a swelling under the tongue. The flow of saliva called forth by the act of mastication is suddenly checked by an obstruction to the salivary

gland, usually by a small calculus or fish bone or other foreign substance, resulting in dilatation of the duct from the oncoming flood of saliva. When the calculus escapes or retreats towards the gland immediate relief and disappearance of the swelling are experienced.

Diagnosis: Ranula takes its name from frog belly—just why I cannot see, as it bears little resemblance to that animal's abdominal region. Dermoids in this region resemble much more the appearance of a frog's belly than do ranulæ. The latter presents a bluish cast, a smooth velvety appearance, not as tense to the touch as a dermoid, somewhat fluctuating, usually located laterally to the frenum, although when large it may present in the median line as well, but then it occasionally is bilobed in appearance from pressure of the frenum. It is confined usually to the region of the mouth, beneath the tongue, and only occasionally presents below the chin into the neck as dermoids do. This is on account of the superficial position of the gland. Its development is fairly rapid, much more so than dermoids, but it never attains to great size, differing in this respect from a dermoid. Its contents is a glairy or mucoid fluid, as a rule clear, at times flocculent, and may even be turbid from contamination. The orifice and main duct are usually patent. On account of lack of resistance met by its growth, it but slightly interferes with the function of the mouth, unless it is acutely inflamed or very large. Sex or age appear to be no important etiologic factor, occurring in male and female alike, children and old age.

Treatment: I have seen a number of true ranulæ, and they were treated by various methods, including incision with evacuation, followed by the application of tincture of iodine, nitrate of silver or trichloroacetic acid and gauze packing. These cases never did well. The incision closed prematurely, there was a reaccumulation of fluid and at times infection. A partial excision of the cyst wall with topical applications brought the case to healing, after a long time and much discomfort to the patient. These cysts are difficult to handle surgically per orum, owing to the adherence of the growth, the interference of the tongue, the bleeding and mouth secretions, and the danger of infection and aspiration. Where this method is followed, the following procedure is advised: Linear incision

of mucous membrane only; careful dissection of same from the sac as far as possible without puncturing; placing a fine wire in a snare holder around the remainder, removing as much of the cyst as possible. After thorough toilet, the remaining cyst wall may be sutured to the mucous membrane edges.

In two cases I followed the advice of von Hippel,¹¹ using the neck incision. Following the lower border of the mandible about 6 cm., dissecting down to the submaxillary gland and displacing it downward, and the digastric, geniohyoid and genioglossus muscles forward, then through the mylohyoid to the cyst in the sublingual gland. This gland is supplied by the submental and sublingual arteries and veins, and these have to be ligatured. A through and through rubber gauze drain is applied for several days, then removed, and in a week or ten days the wound is healed. This method of handling ranula is much preferred to any per oral surgery, especially where a former attempt by incision had been made.

Cysts of the mucous gland more readily respond to the incision or resection method per os than do ranulae, although total excision is at times the only permanent relief.

The epithelial lined cysts include the mucous, dermoid, thyroglossal, colli congenita and the multilocular or adamantinoma. Some of these are simple retention cysts resulting from a closure of the duct's orifice or obstruction within the duct. Some are inclusion cysts resulting from trauma. Others are congenital in origin, as fetal inclusions in foreign soil or fetal rests derived from embryologic clefts, fissures and ducts.

Dermoids of the mouth have received special attention from Gueterbock,¹² Robinson,¹³ Fendt,¹⁴ Klapp,¹⁵ Rocha,¹⁶ Schubmehl¹⁷ and Trumper.¹⁸ Cumston,¹⁹ in reviewing the literature in 1902, found 42 cases. Since then quite a number have been reported. The first recorded case found by Schubmehl was from Jourdain: *Maladies de la bouche*, 1761, and is of special interest, because he attempted to differentiate it from ranula.

My personal observation comprises three cases. A brief note of each case is as follows:

No. 1.—A. K., female, Lithuanian, aged 19, mail order clerk, presented herself on account of difficulty of speech and swallowing. She is the second child in a family of four chil-

dren, three of whom are brothers, all well. Patient had measles and whooping cough only. Examination showed a large symmetric, smooth and yellowish like tumor, occupying the entire floor of the mouth. Its size was such as to push the tongue against the roof of the mouth and to bulge as a globular mass into the neck below the chin. It appeared the size of a small orange. It had a tense but elastic feeling, and was easily movable with the tongue or finger, but not in the least painful. There was no interference with normal breathing, no choking attacks, but some complaint because of difficulty in eating and in talking. It was first noticed when a little girl of five years as a slight swelling below the tongue. Of late it appeared to grow more rapidly, at least it caused greater discomfort from its size, and therefore she presented herself for the first time for examination.

Operation: Cocain locally, then a 1 per cent procain solution submucously. Incision of mucous membrane of mouth to left of frenum, avoiding the salivary ducts. The wall of the cyst was grasped by two forceps and the cyst incised. A mass of pasty clay colored material was disclosed, which proved to be sebaceous. About one-half the contents was easily removed with a large spoon curet, which permitted the removal of the sac containing the remainder through the incision. With slight traction and blunt dissection, the entire cyst was easily liberated, until the pedicle attachment to the hyoid was reached. Scissors were then used to cut this. Two sutures and a drain completed the operation. No complications. Microscopic examination of cyst wall showed connective tissue, pavement epithelium, cylindrical cells, few blood vessels, sebaceous glands, sweat glands and a few muscle fibers.

No. 2.—A. N., female, colored, married, aged 21, housewife, complained of a swelling in the mouth and neck that inconvenienced her in eating and talking, and somewhat in breathing. Had been present and slowly growing for many years. At times she had choking attacks. On examination there presented a large, smooth, round mass, filling the mouth and pushing the tongue upward and bulging below into the neck behind the chin. It appeared and proved to be the size of a small orange. It was elastic to the touch, movable and caused no pain. Skin and membrane normal. The latter had a light yel-

low color, with a few blue veins across the surface. There was no inflammation of the membrane or adenitis present.

Operation: Cocain locally and 1 per cent solution procain submucously. A horizontal incision through membrane in floor of mouth, to left of frenum. The sac was grasped with forceps, then incised and its contents, a yellowish, cheesy mass, removed partly by a large spoon curet and the cyst wall then readily peeled from the surrounding tissue with no bleeding. The firm fibrous pedicle attached to the hyoid was severed with scissors and the cyst removed completely. Two sutures and drain. No complications—excepting a slight delay in healing from accumulation of fluid. Microscopic examination: The contents was sebaceous material; the walls, connective tissue, pavement epithelium, papillæ, sebaceous and sweat glands and few blood vessels.

No. 3.—A young man, aged 20, presented himself, with a history of having a cyst under his tongue for three years. It had been operated on twice, by incision only, but as promptly recurred. It was the size of a marble and caused no special inconvenience. The mucous membrane showed scars of the former incision over a bulging near the frenum linguæ.

Operation: Under cocain and procain anesthesia, and grasping the tumor with two forceps, after incision of the mucous membrane, the cyst was dissected free with scissors and knife and removed. On opening, it contained a light brownish mucoid material and many hairs. Microscopic examination: Fibrous tissue, hair follicles, a few sweat glands and pavement epithelium.

Dermoids are misplaced fetal inclusions that take on activity and form cysts. The sebaceous and teratoid are the two varieties. These are not the same as the acquired type of inclusion cyst arising from traumatic implants, the result of burns, injuries or operations around the mouth. This defect in the orderly embryologic closure of the branchial arches results in retention of the epithelial germs after the ectoderm closes in, and is the reason given by some for the fibrous adhesions to the hyoid and symphysis as indicating the point of entrance. Gerard-Marchant speaks of two kinds, the adgenien and the adhyoidien, according to their adhesions. In the former the fibrous attachment is to the symphysis of the lower jaw, while

in the latter it is the hyoid bone. Rarely are they found free from such anchorage.

Dermoids commence shortly after birth, but on account of their slow and painless growth attract little attention until early youth or adolescence, unless there should be interference with nursing. They project into the floor of the mouth or into the neck behind the chin or both places. At times they attain the size of an orange. Only rarely is there deformity of the jaw or irregular teeth with faulty occlusion. Both the skin and mucous membrane coverings are healthy and normal. They have a pale yellowish cast, feel soft, smooth and elastic, globular in appearance and centrally located. On rare occasions the cyst has been found to the side. Gueterbock reports one found in the left side of the mouth, and Klapp found one on the right side. In both instances they were mistaken for ranula. Their lining membrane consists of connective tissue, squamous or pavement epithelium, sebaceous glands, hair follicles and even sweat glands. Those of epiblastic origin may contain sebaceous substance, hair, epithelial cells and cholesterol. Those originating from hypoblastm contain mucoid fluid and have a connective tissue lining membrane containing cubical and columnar epithelium. In the differential diagnosis, consideration should be given to the possibility of

ranula	suprahyoid bursal cyst
abscess	hydatid
angioma	gumma
hematoma	lipoma
aneurysm	lymphadenitis

Mikulicz's disease

Methods of Operating.—Total extirpation of the cyst is the only method to be entertained. This can be carried out under local anesthesia per os. The sac peels quite readily from its bed, and only its pedicle attachment may cause some delay. This method offers no such difficulties as the removal of a ranula per os. If desired, a horizontal incision in the neck, back of the chin, may be employed, but it is much more of a dissection than the per oral route.

Thyroglossal Cyst.—The work of His²⁰ demonstrated the thyroid gland as developing from three independent parts: the

two lateral lobes and a median tubular part, from the ventral wall of the pharynx, which becomes the isthmus as well as the pyramidal lobe. The tubular is divided into an upper and lower part. The upper is called the lingual duct and extends from the foramen cecum at the base of the tongue to the hyoid bone. The lower is known as the thyroid duct, and extends from the hyoid to the isthmus of the thyroid. This small caliber epithelial lined embryologic structure, known as the thyroglossal duct, can be demonstrated in the embryo of four weeks, after which time it begins to disappear. By the time the hyoid cartilage has developed in the fifth week the duct is obliterated. Should any part remain patent and not disappear, a fistulæ and even a cyst or both may appear later. In certain invertebrates, as the crab and scorpion, it persists, the thyroid being a sex gland and its duct empties into the genital tract. In man a persistence of a portion of the tract is much more frequently found in the infrahyoid segment, either in part or as a whole. C. F. Marshall²¹ found it in 43 per cent of subjects examined, or 60 children, from a few weeks up to 10 years. The suprahyoid segment usually obliterates entirely, although Marshall²² has shown a continuous tract from the foramen cecum to the hyoid. J. E. Frazer²³ thinks some of the cells of the duct may remain and regenerate, although the remainder of the tract has entirely disappeared. A sudden activity after years of quiescence has been attributed to bacterial invasion of neighboring lymph adenoid tissue.²⁴

Cysts and fistulæ of this tract are not rare occurrences. Out of 86,000 consecutive patients examined in the Mayo Clinic, 31 cases were found.²⁵ Out of 117 cases compiled from the records of the London Hospital, Bailey reports 75 occurred in women. Twelve cases have come under my own observation. Six were operated on. Two of the six cases operated on were associated with sublingual cysts. In two of the fistulas the duct was closed at the hyoid, and communication existed only in the throat at the foramen cecum. In the remaining ten cases a fistula in the anterior median neck existed. These external openings result from the rupture of the cyst, and they may be near the thyroid isthmus or in the hyoid region.

The youngest of my patients was four years and the oldest sixty years of age. Seven were females and five males. At

times several members of a family may present similar defects. In one instance three of the children presented sinuses from incomplete cleft closures. All of them had sinuses in front of the tragus. In one there was in addition a sinus in the carotid area, and in another an added thyroglossal fistula with cyst formation. I will briefly mention the two cases operated on for sublingual thyroglossal cysts, because of their interest.

No. 1.—A man, 30 years of age, complained of a "lump" in the mouth that at times became most annoying, because of the limited motion of the tongue interfering thereby with talking and swallowing. On one occasion it was much larger than at any time, creating alarm because of embarrassment of breathing. It was then opened in the mouth by an incision as an emergency measure. The fistula was dry when he first presented himself. A skin scar covered the opening. The size of the "lump," as he called it, varied as to the patency and discharge of a fistulous opening just superior to the body of the hyoid bone. The floor of the mouth showed a smooth, firm swelling near the center, about the size of a pecan. The mucous membrane was not so thin or bluish in color as a ranula, or as yellowish as a sebaceous cyst. There was slight speech defect, as if he had a wad of tobacco in his mouth, and attacks of laryngeal irritation, with a hacking cough which at times caused apprehension for fear of suffocation. It could be felt below the chin by firm pressure in the mouth. No inflammation, no pain. Attempts to establish drainage through the neck fistula by aid of a probe was partially successful, but similar procedure via the foramen cecum brought no result. The injection of methylin blue to determine a patency at this place only caused great pain to the patient and no discoloration in the mouth. An operation under ether anesthesia was performed, using a short collar incision and encircling the fistulous opening. By grasping the fistula, in which a probe had been introduced, in the jaws of a large dressings forceps, and making slight traction, I dissected toward the thyroid, using chiefly a stripping method and occasionally a Mayo dissecting scissors. In this way I readily reached the body of the hyoid. At this point it was difficult to determine whether the fistula passed in front of or behind the bone. It has been reported as passing through the hyoid. Frazer has shown the relation-

ship of the complete infrathyroid tract to the hyoid. The retro-hyoid portion is carried into its position by folding around the lower edge of the bone, giving the appearance of passing through the bone. On this account, he believes that division of the hyoid is seldom necessary. I separated the median raphe of the mylohyoid and geniohyoid, and, at a place where a bulging presented resulting from a finger exerting pressure within the mouth, the sac was uncovered and dissected free by stripping. Its contents were mucoid and its inner wall was mucous membrane epithelium. No thyroid gland tissue was found.

The second case was a woman, 33 years of age. Since childhood, or about 10 years old, she developed a large lump beneath the chin. This very gradually grew larger until one time she put her finger in her mouth and felt the same mass distinctly. Later on she relates a fistula established itself anterior in the neck and the swelling disappeared. Since then it has been discharging. She was under local treatment by several physicians, but the only result was an inflamed skin about the orifice, with destruction of the normal skin, to the size of a thumb nail, and the fistula discharging pus. The opening was near the cricoid and just to the left of the median line. A probe entered two inches upward, inward and toward the right. Injection of a solution of mercurochrome caused pain and swelling. Neither this nor methylin blue passed to the mouth. She was operated on in a similar way as the one just recited. The fistula and sac extending up behind the hyoid was removed in toto, and measured two and three-quarter inches. Its lining was mucous membrane epithelium.

Differential diagnosis: This cyst must be differentiated from a sublingual dermoid, an abscess of a suprahyoid lymph gland, a suppurating infrahyoid bursa and from accessory thyroid substance. The ingestion of food has been observed to create an increase in the amount of discharge from a fistula or an increase in the size of the cyst.^{26 27} On protrusion of the tongue the cyst is pulled upward, receding as the tongue is withdrawn.

Under colli congenital or branchial fistulae one understands an embryologic deficiency of development in the neck, the result of a persistent cervical sinus. They show themselves in three places, along the outer side of the neck, anywhere from

the mastoid tip down to the head of the sternum. The other two places are in the throat, in the region of the tonsil and in the region of the sinus pyriformis. Where the branchial cleft closes, both internally and externally, but an intermediate portion remains unobliterated, the lining epithelium may proliferate and by other changes create a cyst. These cysts usually occupy the submaxillary region and rarely involve the floor of the mouth. Occasionally such a cyst may grow so as to encroach upon the floor of the mouth from below the mandible, as is shown in a patient who came under my observation. My patient was aged 55, well proportioned, healthy otherwise, the only complaint being the unsightliness of a large swelling in upper angle of right neck. It was first noticed by patient thirty years before. Its growth was slow and painless. On examination it was the size of a large hen's egg, immovable in the neck, but a swelling in the floor of the mouth of the same side could be depressed with the finger and showed to be part of the neck lesion. There was neither skin nor mucous membrane changes, no pain, no inflammation and no adenitis. The dissection necessary for its removal extended into the sublingual field, the specimen proving to be a compound cyst involving the cervical sinus and the submaxillary gland.

J. E. Thompson has observed several such compound cysts, and he believes direct relationship exists between ranula and branchiogenetic cysts.

The multilocular cyst of the jawbone is one of the least frequent of all. It is known as cystic adenoma or adamantinoma. It is easily confused with sarcoma, although benign in nature. Malassez describes it originating as epithelial rests derived during fetal life from (1) mucous membrane of the jaw, (2) from epithelial cord of enamel organ, (3) from epithelial membrane of enamel organ. It encroaches on the floor of the mouth by an extension inward from the mandible. It can scarcely be confused in diagnosis with any other variety of cysts found in the mouth. First, because of the deformity of the jaw; second, because it is integral with the jaw itself and hence immovable, and third, because of its firm bony feel.

The echinococcus cyst, although of common occurrence elsewhere in the body, only rarely is found in the mouth. It presents as a chronic cystic enlargement, causing mechanical in-

convenience alone, and no pain, unless it should become infected and suppurate. When in the mouth they are usually of the tongue.

SUMMARY AND CONCLUSION.

Laryngologists occasionally encounter cystic formations in the floor of the mouth that require a differential diagnosis because of the different methods of procedure followed in their removal.

Several varieties of cysts occur in this region, among which are ranula, dermoid, thyroglossal, mucous, branchial, multilocular and echinococcus.

A histopathologic study of their walls has given an accurate understanding of their origin and structure, and this knowledge has lent itself to the construction of a symptomatology and clinical picture which permits of a correct diagnosis and method of operating.

The laryngologist is now equipped with the information necessary to handle these cases successfully, and he should develop his ability to a greater extent so as to successfully cope with this class of surgery by eliminating infections and lessening complications and recurrences.

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LXXXIII.

RESULTS IN RADICAL MASTOID OPERATIONS
AS TO HEARING.*

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Successful as we may deem the radical mastoid operation from a surgical standpoint, how do the results appear to the patient? The fact that he is rid of a discharge from the ear which has long ceased to trouble him does not appeal to him as strongly as the potential change in his hearing. He is keenly interested in learning as to the possibility of improved audition. What can we tell him? How far are we justified in holding out to him the prospect of increased hearing?

The records of large institutions where many such operations are performed afford examples not only of the brilliant results which may be obtained, but also show an almost discouraging list of failures. Often where the surgeon has achieved a complete success, and the ear is free from discharge, the hearing is practically nil. Fortunately there are many cases with more gratifying results. An attempt will be made here to enumerate certain of the causes, anatomic, clinical and pathologic, which of necessity are important factors in conserving the hearing.

We all know that there must be no impediment to the vibration of the ossicular chain in the transmission of sound waves to the auditory nerve. The breaking of this chain by removal of the drum membrane or one or more ossicles does not present an insuperable barrier to such transmission. Adhesions binding the drum or any member of the ossicular chain may, however, effectually prevent useful hearing. Why? Without movement of the perilymph, there can be no stimulation of the auditory nerve. Fluid is incompressible. Block one of the

*Read at the fifty-eighth annual meeting of the American Otolological Society, May 5, 1925, Washington, D. C.

windows opening into the vestibule, and there can be no compensatory movement allowing free vibration of the perilymph. Free this window, and hearing will be restored. This applies to either the oval or the round window.

Bárány once tried an interesting experiment on a man with an immovable stapes who suffered great deafness and tinnitus. Under local anesthesia the mastoid was opened, the posterior semicircular canal exposed, and a small window cut through, exposing the membranous canal. Immediately the patient heard! This proved that hearing depended upon compensatory vibrations in the labyrinth. This same result was achieved in another manner on many occasions by Dr. Jack. He resorted to the expedient of removing the stapes, reporting satisfactory results. Removing the immobile obstruction to the oval window, together with a normal round window, accomplished the desired result.

The question of hearing following removal of diseased ossicles was painstakingly investigated by the late Dr. J. Orne Green. He found that there was frequently a considerable degree of hearing remaining following the removal of the malleus and incus. His patients were subjected to a long course of postoperative treatment, in which the daily use of the middle ear syringe and the instillation of a solution of boracic acid in alcohol played a prominent part. No work was done on the tensor tympani muscle, except a simple tenotomy, and no attempt was made to clear the eustachian tube. Such granulations as could be removed were taken out, and, if difficult to remove, a silver bead was used freely.

The important fact is that these cases got rid of their supuration. They also improved in hearing. Under this intensive treatment the membrane on the inner wall of the middle ear either epidermatized or cicatrized. But it remained a thin membrane, without the large masses of organized granulations often seen in radical mastoids today. This was undoubtedly an important factor in the improvement of hearing. Several of the members of this society were privileged to follow this work of Dr. Green's, and can bear witness to the truth of the assertion that not only were the results as to dry middle ear and hearing good but in many cases lasting.

Now in the radical operation, we but ream out the mastoid and connect it to a middle ear cavity prepared something after the fashion of the ossiculectomy just described. Why, then, should we not achieve at least as good a result as to hearing? Minute examination of the promontory in healed radical mastoids often fails to reveal the stapes, or round window. In fact, there are few recognizable landmarks. Instead of the familiar anatomic markings of the promontory and windows, we are apt to find in the middle ear a smooth impression, as if the end of the finger had modeled the cavity out of wax. This is where there is no moisture. In other cases there may be granulations and discharge at the site of the eustachian tube and hypotympanum. It is all too apparent that granulations have encroached upon the middle ear, and have organized and become epidermatized. By covering the oval and round windows, they have as effectually sealed the organ of hearing as the eruptions of Vesuvius did Pompeii.

The methods at our command to combat this encroachment of granulations and connective tissue on the middle ear with practical obliteration of the cavity come more properly within the scope of one of the other papers to be read this morning. But skin grafting alone will not necessarily lead to a successful result—at least as far as function is concerned.

To recapitulate: We have shown that following simple ossiculectomy ears may become permanently dry with good hearing. With more extensive operation the results are often not as favorable. The writer believes this to be due to reliance upon means for healing the mastoid cavity, and to permitting the middle ear to care for itself. The view has often been advanced that poor hearing following the radical operation is due to the placing of a skin graft over the stapes. This view does not seem tenable, for several reasons. The ordinary graft, applied in any manner, simply rests over the pelvis ovalis and possibly touches the head of the stapes. It does not descend into the oval window. In due time it sloughs off, leaving the new cells to spread. They quickly form a thin, dry epidermis, protecting both windows. This is the most favorable condition, and is exactly analogous to what transpired following the earlier ossiculectomies, although possibly the graft facilitates the spread of epidermis. It would seem that the only way in which

the insertion of the graft could do harm would be by covering up granulations in the middle ear with later formation of connective tissue or by using a tremendously thick graft. In the latter event a successful take would mean the obliteration of the middle ear, with dire results to hearing.

Much more common in the unsuccessful cases that have come to the observation of the writer are the ones where a raw, discharging surface persists in the middle ear. The following case illustrates this type:

Case 1.—Female, 40, had abscesses a. u. in childhood. There were scars and perforations in both ears, but they had healed. In 1913 she had inflammation of the left middle ear, followed by mastoid operation. After recovery the hearing remained unchanged—very poor a. u. She remained well for nine years, when the left ear again discharged. It did not yield to treatment, and during the next year the onset of severe headaches in the temporal region decided us to perform the radical operation. This was in January, 1923. To this day the writer is ignorant of any cause which should have led to failure, but fail the operation did, and most ignominiously. To be sure, the posterior wound healed, but the primary graft did not take. Granulations filled the mastoid and tympanic cavities, and worse than all, the canal gradually grew smaller and smaller. Six months after the operation it was almost impossible to see the region of the middle ear. Moist granulations filled the entire cavity. There was no particular change in the hearing. Low conversational voice could be heard at one foot. As the results were so bad, the patient, somewhat to my surprise, was eager that another attempt be made to improve the condition. In October, 1923, nine months after the radical operation, the wound was again opened. Granulations throughout the cavity were carefully removed. It was not necessary to change the bone cavity in any way. A primary skin graft was again inserted. To the amazement of all concerned, the wound healed promptly. In three weeks there was no vestige of moisture in any part. New skin covered the entire inner portion of the mastoid and middle ear. Best of all, the hearing, which had been poor, to my personal knowledge, for twenty years, suddenly improved from ability to

hear conversation at one foot to hearing with ease at twenty feet. This improvement has persisted.

In this case it is obvious that the sound perceptive apparatus had remained unimpaired many years. Clearing away debris in the middle ear was all that was necessary to restore good hearing.

These additional cases are presented in support of the claim to good hearing following the radical operation. All, including the one just given, are from private practice.

Case 2.—Female, 44. Earaches in childhood. Onset of suppuration a. d. three years ago, without pain. Continued despite prolonged treatment by competent aurists in various cities. Radical operation a. d. March 19, 1921. Primary graft. Hearing before operation a. d. low voice, one foot; whisper not heard. Last seen one month ago. At that time, ear dry, no crusting or discharge, hearing a. d. whisper eight feet, low voice twelve feet.

Case 3.—Male, 30. Chronic abscess a. d. all his life. Is a trial lawyer and finds deafness hampers court work. Radical mastoid operation in June, 1906. Secondary graft. Unable to hear whisper or voice a. d. before operation. Have seen patient at intervals of one or two years during intervening nineteen years. Last seen April 1925. Condition then, a. d. dry with small amount of epithelial desquamation. Hears whisper two feet, low voice ten feet. Entirely satisfied with result of operation.

Case 4.—Female, 8. O. m. s. ch. a. u. since two years old. Prolonged treatment cured discharge in a. d. Radical operation a. s. in November, 1905. Secondary graft. Heard loud conversation only in either ear when first observed. Last seen in February, 1925. Both ears dry. Unoperated ear practically useless. The left, the side on which the radical operation was performed twenty years ago, heard low conversation easily at ten feet. Patient is a school teacher and has no difficulty in hearing pupils.

Case 5.—Male, 7. Scarlet fever when two years old. Emerged from this nearly blind and so deaf that nothing less than a shout could be heard. There was chronic discharge and

granulation tissue present in each ear. Patient so helpless, both blind and deaf, that the parents were willing to go to any extreme to aid him. Right radical operation in January, 1919. Primary graft. Healed in one month. Could then hear low conversation at three feet a. d. The result was so good that it was decided to operate on the left ear. This was done four months later. Radical operation left ear, primary skin graft. Healed in about the same time as the right, one month. Result as to hearing not quite as good as with the first ear, but repeats low conversation at two feet. Last seen in September, 1924, five years after operation. Both ears have remained dry and have retained the gain in hearing.

Case 6.—Male, 13. First came under my observation when two years old. At the age of five months had mastoid operation a. d. Brought to me because he did not talk. Both drums much retracted. Could not be sure that he heard any sound. Tonsils and adenoids removed, and ears inflated for a spell. As the boy grew older, and still did not talk, he was placed in charge of faithful tutors, who labored patiently with him for some years. The family then moved to California, and I did not see him again until he was thirteen. In the interim he had developed on abscess a. d., which did not yield to treatment. He had learned to talk, but could hear only with the greatest difficulty. Bone conduction was such, however, that it seemed justifiable to operate on the right ear, with the hope of improving the hearing. The radical operation was performed in June, 1919, and a primary graft used. The ear was healed in three weeks, and at the expiration of the month he returned to California. Reports were satisfactory. I did not see him again until June, 1923, four years after the operation. A marvelous change had come over him. He heard so well that he had completed his freshman year at Leland Stanford, and was looked upon as a student of great promise. All tuning forks were heard by air in the operated ear, and low voice easily heard at ten feet.

Case 7.—Female, 22. Abscess left ear when six years old. More or less discharge since; worse past two years. There was a perforation in the lower posterior part of the left m. t., with foul discharge. There was inability to hear the voice at all in the left ear, and a negative Rinne. After personally

treating the ear for six months, radical operation was resorted to in October, 1916.

A skin graft was inserted a week later. This case did not heal well. Granulations kept reforming in the middle ear, and it was not until February, 1917, that the ear became dry. This was four months after the operation. At that time she was able to hear low conversation six feet in the operated ear, and shortly after entered the U. S. Navy as yeoman (F), passing the medical examination without difficulty.

Case 8.—Male, 30. First came to me in September, 1906. Had been a patient of the late Dr. J. Orne Green. History up to that time o. m. s. ch. a. u. past fifteen years. Ossiculectomy a. s. twelve years ago. Mastoid operation, left side, two years ago, by Dr. Green. Both ears continued to discharge. Two weeks before this recent history, had nausea and vomiting for days. No chills. At this examination there was considerable swelling in the left auditory canal, with granulations in the attic, and a most foul smelling discharge. He was placed in a hospital, where the granulations were removed, but no major operation performed. He recovered from his symptoms and disappeared from observation. It was not until ten years later that I next saw him. This was in June, 1916. He then came with the story that the left ear had been discharging more than usual for a month past, and that there were pieces of cholesteatoma coming from the ear. The day before he had had slight headache and some nausea. No vertigo. Reflexes normal. Temperature 99. Pulse 84. He again went to a hospital for observation. The symptoms, other than the suppuration, soon cleared up. As he had had several attacks with bad head symptoms, it was finally decided to perform a double radical mastoid operation. This was carried out in September, 1916, both sides being operated upon the same morning. Secondary skin grafts were applied the following week. He left the hospital in two weeks, and was healed in six weeks. This patient has been under my observation at least once a year since. His ears have remained dry. There has been no recurrence of headaches or vertigo. He could not hear conversation in either ear before the operation. For the past nine years he has been able to converse with ease, even with strangers, in an ordinary room.

There are many more cases, equally fortunate in outcome, in the list from which these were taken. We all have them. We all, probably, have our bad cases. I, at least, have had my share. But as I ran over the list of patients who had undergone the radical mastoid operation, it seemed distinctly worth while to place emphasis on the fact that these cases get well, remain well, and frequently maintain or recover a surprising degree of hearing.

LXXXIV.

VACCINES FROM HEMOLYTIC COCCI IN SPHENOTHMOIDAL DISEASE.*

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Following the false dawn of vaccine therapy in 1895¹ and the temporary and unauthorized excitement over Marmorek's work with the streptococcus, a veritable sunburst of enthusiasm for vaccines again occurred about twenty years ago. Hasty proponents of the new serologic vocabulary were busy with papers and case reports; and the pharmaceutical houses, with biologic departments and detail salesmen, gave practical emphasis to Shakespeare's

"Our remedies oft in ourselves do lie!"†

This association was among the first to take note of the trend toward bacteriologic investigation and therapy of the accessory sinuses, following Boucheron's pioneer case in 1897.² The admirable essay of Hinkel,³ in 1902, on chronic sphenoiditis, mentions the continued menace of mucopurulent pharyngeal discharge, but does not suggest vaccine treatment. Not until 1910 did the association invite general discussion of this method. Birkett and Meakins⁴ then claimed that, with vaccines based mostly upon the staphylococcus aureus, they had been able to sterilize the accessory sinuses; their recommendation was mainly for subacute or chronic cases, following operative procedures. Coakley and Kendall⁵ took a contrary view, stating that the opsonic index and the vaccines were useless in their cases, save for one sphenoid and one antrum infection of streptococcic origin. Cobb and Nagel⁶ had nega-

*Read before the annual meeting of the American Laryngological Association, Washington, May 4-6, 1925.

†"All's Well that Ends Well," Act I, sc. 1.

tive results. The following year Casselberry⁷ entirely omitted vaccines from his treatment for suppurative sphenoiditis. But Coffin,⁸ in 1915, voiced a certain discontent with results, even after the most thorough and radical operations; he counseled autogenous vaccines, along with suction and oily antiseptics. First mention of the hemolytic streptococcus was made by Halsted⁹ in 1917, discussing cases of general infection of accessory sinus and tonsillar origin.

In other societies similar discussions prevailed; enthusiasm at first, then general use of autogenous or stock vaccines, and a waning interest after two or three years, relegating vaccines to the cloistered retirement of a few "selected cases." Coates¹⁰ was among the earliest to emphasize that vaccines had no place until necessary surgery had been done, and,¹¹ in 1917, pointed out the fallacies in missing true toxic agents, due to faulty cultural methods. His results were good in preventing recurrences. Levy¹² had postulated, in 1909, that all obstinate cases in which other methods had failed should have the benefit of vaccine; he reported 6 cured, 6 improved, 3 unchanged. Patterson,¹³ in 1910, did not mention hemolytic organisms, nor did he employ the streptococcus. Brawley's¹⁴ best results were in cases not over a year's standing. Graef and Wynkoop¹⁵ in the same year reported a brilliant cure of streptococcic meningitis after a septum operation, by autogenous vaccine. Dabney's¹⁶ vaccines in 1912 were made up in the proportion of one part streptococci to ten of staphylococci, without hemolytic differentiation. Haskin,¹⁷ in 1916, was first to use an autogenous vaccine from streptococcus hemolyticus, in a case of nasal and oral pemphigus.

In spite of wide interest throughout the country in these results (See Bibliography, 18 to 24), and more recently abroad (See Bibliography, 25 to 34), and in spite of the propaganda of stock vaccine salesmen, rhinology has turned away from the use of vaccines. Skillern has well stated the prevailing pessimism, not only in his treatise,³⁵ but more recently in his masterly review of the history of the sinuses.³⁶ Mosher,³⁷ nevertheless, directs attention to the probability that toxins may be elaborated in the sinuses without apparent pus, and even without appreciable bacterial growth.

Addressing ourselves to the bacterial side of the question, we are reminded that vast changes have come into the culture and identification of bacteria during thirty years; and in corollary thereto, that the entire theory of cell chemistry and of protein sensitization and therapy is now under active revision.

Stock vaccines, as well as polyvalent autogenous vaccines, may well be dismissed from present consideration. Their value, because of multiple protein content, can scarcely be specific in any case, and probably depends upon the stimulant action of foreign protein upon the blood and its defensive mechanisms.

It is folly to assume that casual swabbing or washing from any nose or throat will even approximate the percentage of pathogenic germs existing thereafter. Only pure or highly preponderant cultures should be considered. Here again is room for fallacy; on certain media weedlike growth of non-pathogenic germs speedily obliterates the dangerous species. Skilled differential culture is needed. Such cultures should be repeated at intervals during the course of each case. Growth upon the subject's own blood is favored by Solis-Cohen.⁵⁸

Injection of alien protein, either milk (conceded by many to owe its revulsive value to its variable content of bacteria), or diphtheria antitoxin,³⁸ or typhoid vaccine,³⁹ or bacillus coli vaccine,⁴⁰ or plain horse serum, has been employed for some years against chronic ocular inflammations. Duke⁴⁰ uses it in asthma and hay fever. Rather violent temperature and skin reactions accompany the leucocytic increase in the blood after use of these agents, whose discomforts and dangers are not to be ignored.

Infestation of the population with streptococci and other hemolytic organisms has been a noteworthy problem in epidemiology during recent years. Bacteriologists have discovered a considerable proportion of hemolytic germs in cultures from the nose and tonsils.^{41 42 43 44 45} Some observers have inclined to the belief that filterable virus from unrecoverable organisms is to blame for acute colds and for lighting up chronic disturbances. Others depend upon improved cultural methods, animal inoculation and clinical experiment for determination of specific infective agents.

Every rhinologist will admit that he sees certain cases of sphenothmoidal disease which seem to resist every type of treatment; cases which too often wander from one man to another, while we invoke

“ . . . a kind of hope
Which craves as desperate an execution
As that is desperate which we would prevent.”*

Here are found the asthmatic types—protein allergies, of course, excepted; sphenopalatine neuralgias; relapses after complete operations; inoperable cases; pregnant women, marantic children, the aged; diabetics, albuminurics; the high anemias and hemophilia; septicemia; heart cases; the tuberculous. Free pus is often not found;^{37 58} telltale lymphatic reddening behind the posterior pillars, or burning sensations, with dry, intractable cough, may be the only sign.⁵⁸ The roentgen ray will show clear sinuses; and if opened, their walls will often show but slight congestion, and a little pale mucus which is sterile to ordinary culture media.³⁷

Methods of obtaining material for the fine differential technic of the bacteriologist have often been excessively crude. Brawley¹⁴ used irrigation and aspiration. Skillern³⁵ advises a sterile sound inserted into the sinus: a difficult problem sometimes, unless much preliminary surgery be done. Ordinary cotton swabs pick up a freight of vestibular mucus coming and going, although much work has been done in this way.^{44 58} The West tube, passing into the mouth behind the soft palate, is complex and easily contaminated, although recommended by Pilot and Pearlman.⁴⁵ These investigators have accurately studied the shortcomings of cotton swab cultures. Others are making use of washings from various sinuses, notably antrums and frontals.

For our investigation, cultures were made, with a long platinum loop set in a glass rod, inserted through a long bladed speculum, after preliminary vestibular cleansing, flat-wise across the middle turbinate into the sphenothmoidal recess. Loop and rod are flamed clean after each loopful is inoculated.

*“Romeo and Juliet,” Act I, sc. 2.

Under the advice and direction of Dr. Robert L. Benson, head of the Department of Pathology, University of Oregon Medical School, two sorts of media were inoculated from each nostril of each case. Without Dr. Benson's special and painstaking skill, this study would have been impossible, because of the faulty technic which impairs the accuracy of ordinary clinical laboratories. The first medium was 1 per cent dextrose beef-infusion agar, to which citrated human blood was added, in the proportion of 1 cc. to the ordinary agar slant. The second medium was 1 per cent dextrose beef-infusion broth, to which 1 cc. of citrated human blood was added, as each culture went into the incubator. Plating out of the organisms was also done on human blood agar. Final determination in cases of doubtful hemolysis was in all cases made on blood infusion agar without dextrose.

A few experimental cultures convinced us of the frequency of hemolytic organisms in the sphenothmoidal region of such uncured or inoperable cases as were mentioned. This important factor in pathogenicity, the power of a given organism to attack and disintegrate the red blood corpuscle, discoloring and destroying hemoglobin, and with it the power of oxygen fixation, was first mentioned by Marmorek for streptococci in 1895;⁴¹ and by Kraus,⁴⁶ in 1900, for staphylococci. Hemolysins are filterable substances, "produced in quantities roughly proportionate to the virulence of the particular microorganism." "Absolutely avirulent races do not, so far as we know, produce hemolysins." (Zinsser.⁴⁷)

Several types of hemolysis have been worked out, following the work of Smith and Brown⁴⁸ and of Benson and Sears^{49 50} on streptococci isolated from milkborne epidemic tonsillitis. With streptococci, the alpha type has feeble, indefinitely outlined 1-2 mm. zones of discoloration about each colony. Type alpha¹ has a sharply marked discolored zone resembling the clear, transparent, completely hemolyzed, colorless 2-4 mm. zone of type beta. The former is never pathogenic, however;⁵¹ the beta type practically always so. For the purpose of our clinical series, no hemolysis, either streptococcic or staphylococcic, was considered unless it conformed to the beta type. Following is a table of all such organisms found in thirty of a total of fifty cases:

TABLE I.

	Times found	Per cent
<i>Streptococcus hemolyticus</i> , long chain	5	10
<i>Streptococcus hemolyticus</i> , short chain.....	7	14
<i>Staphylococcus albus</i> (hemolytic).....	14	28
<i>Staphylococcus aureus</i> (hemolytic).....	24	48
<i>Streptococcus viridans</i> (green pigment).....	2

Two highly anemic cases showed the *streptococcus viridans*, which is feebly hemolytic, with formation of a green methemoglobin pigment. Nearly half of our hemolytic cases showed *staphylococcus aureus*, an interesting parallel to the work of Birkett⁴ in 1910.

We have felt that hemolysis, when found in relatively pure culture organisms, would afford us a notable criterion of general damage which might be suffered by the individual so infested. Cognizance of this fact has recently been taken not only by workers on the rôle of soluble filtrates in epidemiology, and other bacteriologists,^{47 52 53 54} but also by internists considering the etiology of pernicious anemia,⁵⁵ as well as of visceral disease.⁵⁸

Cultures taken in our series of fifty persons presenting the various indications already stated were positive for hemolytic organisms in the sphenoethmoidal region in thirty individuals, or 60 per cent. In eight cases, 16 per cent, it was necessary to repeat the culture once, and in two cases, or 4 per cent, twice, to secure a practically pure or heavily predominant culture of the suspected organism. In one sided lesions the predominant organism was isolated from the affected side in over 90 per cent, but in only about 60 per cent of simultaneous cultures from the uninvolved side—a point suggestive of the tissue resistance inherent in undamaged mucosal surfaces.

The average age of these patients was 31 years and 5 months; of six children under 12, the youngest was 7; of four adults past 45, the oldest was 71. There were nineteen males and twenty-four females in the series treated by vaccines or proteins.

Ten cases, 20 per cent, disclosed a predominant nonhemolytic *streptococcus*, from which vaccines were prepared and used

as controls. Three cases, 6 per cent, presented asthmatic symptoms without hemolytic organisms; these three were treated as controls, with typhoid vaccine. The remaining seven cases, 14 per cent, presented neither streptococci nor hemolytic organisms, and were not considered further for vaccine treatment.

All cases were private, insuring more accurate control of dosage, reactions, and details of results.

Vaccines were prepared in the concentration of two billion killed organisms to the cc., and preserved in a cold place. Contrary to the practice of ten years ago, when 0.5 cc. was the usual starting dose for a vaccine containing four to six kinds of killed bacteria,¹⁰ we began with an average dose of 0.1 cc., and in some cases, as children or prostrated invalids, used as little as 0.05 cc. to start. Table 2 shows the initial reaction to each type of vaccine, by percentages.

TABLE 2.
INITIAL REACTION TO VACCINES.

Kind of vaccine	Slight	Moderate	Severe	Excessive
Hemolytic	28%	28%	42%	2%
Nonhem. strep.....	10%	30%	60%
Typhoid	25%	75%

Such vaccines were given every four days, injected subcutaneously, preferably in the back or over the shoulder blade, after alcohol sterilization. Subsequent doses increase 0.1 cc. each time unless considerable general reaction occurs, in which case the previous dose is repeated. On reaching the tolerance limit, usually 0.3 cc. in children, rarely over 0.5 cc. in adults, injection is made weekly. Local reaction was noted but twice, an indurated wheal which disappeared before the next hemolytic injection date. Two of the cases of typhoid vaccine control had large painful lumps after 0.5 cc. injections; these disappeared slowly, as commonly with this agent. One case, apparently cured by vaccine of a rather severe recurrent nasal erysipelas, was exposed to typhoid through milk, and received the usual typhoid vaccine prophylactic from his family doctor. Terrific temperature reaction was immediately fol-

lowed by a most violent erysipelas of the whole face and scalp. This case is highly suggestive of the rôle played by alien proteins, whether pollens or food allergies, or animal exudates, in lowering the threshold of resistance to organisms previously well tolerated in the nose and throat.

Because the nasopharyngeal flora changes rapidly during the process of immunization, repeat cultures were taken in many cases, so that 47 hemolytic vaccines were used in the 30 cases analyzed. Dr. Benson properly insists that success in the use of these vaccines can only be reached by keeping the vaccine abreast of the nasal infestation at any given time. Bacteriology is constantly developing new variants in the growth, affinities, preferences and metamorphoses of various strains of the cocci. It may readily be assumed that an immunized individual may develop high resistance against one type of organism, thus permitting another to crop out and be isolated at subsequent culture dates. The fallacy of stock vaccine therapy, save as it is used as a somewhat unstandardized method of alien protein injection, is too apparent to demand refutation at this time. Even as alien protein, stock vaccines have many of the objections incurred by whole milk injections—multiple protein content, and unknown proportions of organisms of varying serologic potency. Typhoid vaccine, containing but one well studied organism, affords an accurate standard for control work on alien protein; and it does not contain the soluble filterable hemolysins which might assist the action of a mixed coccus vaccine.

Table 3 shows the clinical diagnoses in the series:

TABLE 3.

CLINICAL DIAGNOSES IN FIFTY CASES.

Diagnosis	Hemolytic cases	Non- hem. strep.	Typhoid
Ethmoiditis, one side.....	7	1	---
Both sides	23	9	3
Sphenoiditis, one side.....	2	---	---
Both sides	3	2	1
Maxillary, one side.....	4	2	1
Frontal, one side.....	1	---	---

Operative procedures which had preceded the use of vaccines were as follows:

TABLE 4.

PREVIOUS OPERATIONS IN FIFTY CASES.

Operation	Hemolytic cases	Non-hem. strep.	Typhoid
Middle turbinate, one	1
Both	2
Ethmoid, partial, one.....	3
Both	4
Ethmoid, complete, one.....	3
Both	4	2
Sphenoid, one	3
Both	3	2
Maxillary, one	2	1
Septum, partial.....	1
Complete	6	3	2
Tonsil	16	3	2
Adenoid	9
Frontal, internal.....	2

It should be noted that sphenoid, ethmoid, turbinate or septum operations had been done on 24 of the 30 hemolytic cases, and on 5 of the 10 nonhemolytic streptococcus controls. Most of these results were excellent as to improvement of drainage; a few had small foci which required attention before vaccines could be considered. Most of the surgery had preceded vaccine work by periods ranging from several months to ten years. Recurrence of former symptoms was usually the reason why operated cases sought further treatment. In most instances strong objection was made to any more operative procedures; occasionally operative procedures were blamed for exacerbation of symptoms. Our conservatism regarding surgery in such cases was strengthened by the views of Mithoefer,⁵⁶ who decries operation in the presence of hemolytic organisms; and of Shambaugh,⁵⁷ who is averse to operation on purely neuralgic indications, especially where true suppuration is undemonstrable. Pathogenic selective culture, as advised

by Solis-Cohen,⁵⁸ for the individual might be desirable in such cases.

Five of the 30 hemolytic cases were highly sensitized to a large number of proteins, as also were one each of the non-hemolytic streptococcus group of 10 and the typhoid control group of 3.

The degree of anemia in these persons was as follows:

TABLE 5.

ANEMIA AS AN INITIAL FACTOR IN FIFTY CASES.

Type of case	Anemia, marked, %	Moder- ate, %	Slight, %
Hemolytic cases	21	45	34
Nonhemolytic controls.....	20	20	60
Typhoid controls	33	67	...

Complications of these cases included the following conditions:

TABLE 6.

COMPLICATIONS EXISTING IN FIFTY CASES.

Condition	Hemolytic cases	Non- hem. strep.	Typhoid
Pregnancy	2
Dysmenorrhea	3	1
Metrorrhagia	3
Arteriosclerosis	3
Hemophilia	1
Mitral regurgitation	2
Mitral stenosis	1
Myocarditis	2
Septicemia (positive culture)	2
Asthma	10	1	3
Bronchitis, chronic.....	2	1	2
Mucous colitis.....	2	1
Arthritis, acute.....	1	1
Arthritis, chronic.....	3	1

Following is an analysis of symptomatic results in cases treated with hemolytic vaccines:

TABLE 7.

SYMPTOMATIC RESULTS, THIRTY HEMOLYTIC CASES.

Symptom complained of	Cured	Improved	Relapsed	Unchgd
Asthenia	3	4	---	---
Nasal discharge	10	3	1	2
Nasal obstruction.....	8	---	1	---
Dry burning throat	3	1	---	1
Intractable cough	6	2	---	1
Asthma	7	1	---	2
Low fever (99-100.6)	6	1	1	---
Headache, frontoparietal.....	3	---	1	1
Headache, occipitomastoid	8	1	---	1
Recurrent colds.....	5	2	---	---
Recurrent erysipelas	1	---	---	---

Obviously certain of these cases were unfit for further operative procedures, aside from their personal objections to methods tried before and found wanting. Most remarkable were the symptomatic results in two children thought to have whooping cough because of the character of the paroxysms. Cessation of cough after one or two injections was a surprise alike to us and the attending pediatricians. One case of malignant endocarditis at the mitral, bedfast for months with a fever from 100° to 101°, who had previously had an extensive ethmoid well done on the affected side, was after two injections free from fever, and in a month able to sit up and even move about her room. Most of the cases required long continued and careful work, however.

In the following list, the disappearance of all symptoms for four months or more following cessation of the use of vaccines is reported as cure. Prolonged freedom from more than half of the symptoms is reported as improvement. Two-thirds of the hemolytic series have been well for periods ranging from six to eighteen months; the rest have maintained cures or improvement in spite of severe winter conditions. It is, of course, impossible to say whether some may not relapse, and records will be kept to check all these results. Numerical list of cases is appended to the paper.

TABLE 8.

GENERAL RESULTS OF VACCINES.

Vaccine used:	No. of cases	Im'diate cure, %	Cure in 4 weeks, %	Late cure, %	% Im-proved	% Un-changed
Hemolytic (47 vaccines)	30	13	37	23	17	10
Nonhemolytic streptococcic	10	..	20	10	40	30
Typhoid	3	100

The original infective agent was found to have disappeared in every case where final culture was made in the hemolytic series. Several cured cases returned at the beginning of their second winter and requested cultures so as to be protected by vaccine against recurrence; and in a few cases hemolytic organisms were found in small numbers in these persons, and prophylactic vaccines were made.

CONCLUSIONS.

1. Mixed vaccines with numerous organisms, used in sphenoethmoid disease, depend largely upon somatic reaction to alien protein for such value as they may possess. Their effects are likely to be dangerous and are undefinable in proportion to their dosage of different bacteria, each with its own coefficient of protein reaction.

2. Alien protein injections, if used, should be made with some highly standardized product, such as typhoid vaccine. This product was found to have no beneficial effect in three control cases of our series.

3. Autogenous vaccines from hemolytic cocci secured by loop inoculation on human blood media were found curative in 73 per cent of cases of obstinate sphenoethmoid disease.

4. Autogenous vaccines from nonhemolytic streptococci, secured by loop culture on human blood media, were found curative in 30 per cent of cases of obstinate sphenoethmoid disease.

5. Injection of vaccines based on hemolytic cocci is recommended and was tried only upon such cases as were unsuitable for, or had failed to respond to, the established procedures of surgical and antiseptic treatment.

Grateful acknowledgment is made to Dr. Ross H. Skillern for suggestions regarding this study; to Dr. Robert L. Benson,

for invaluable assistance and advice, in preparation of the whole series of vaccines; and to my associate, Dr. Irving M. Lupton, for data on numerous control cases.

May I express the hope that this preliminary report, based on fifty cases observed from 1922 until the present, but requiring confirmation of results over a period of years, may persuade others with greater clinical opportunities to assist in the ultimate decision on the value of hemolysis as a criterion for the clinical usefulness of vaccines? To quote from Marmorek's classic study of the streptococcus:¹ "Nous n'ignorons pas combien la prudence est nécessaire quand il s'agit de juger un remède nouveau. Nous nous garderons donc de toute conclusion hâtive." (We have not forgotten how necessary prudence becomes when the question of judging a new remedy arises. We shall therefore be guarded against all hasty conclusions.)

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HEMOLYTIC SERIES:

No.	Name	Age	Sex	Diagnosis	P Indicates Prophylactic Vaccine	Organisms Found	Vaccine begun	Dose, Max. cc.	Vaccine ended	Result
1.	M. N. J.	32	F.	Eth. and sph. B., opt. atroph., streptococcemia, hemophilia		1. Strep. short ch. 2. Staph. aureus	11/27/22 1/28/24	0.1-0.4 0.1-0.5	1/27/23 3/14/24	Impr. Cure
2.	H. G. P.	56	M.	Eth. L., recur. erysipelas (recurrence after typh. vacc. 1/24)		1. Strep. long ch. and staph. aut. 2. Staph. aut.	10/29/23 12/14/23	0.1-1.0 0.1-0.4	12/11/23 12/23/23	Impr. Impr.
3.	G. H.	16	M.	Eth. B., acne glossitis		1. Strep. long ch.; strep. short c.; staph. aureus. 2. Staph. albus.	8/ 6/24 1/ 7/24	0.1-0.5 0.1-0.5	10/18/24 2/ 5/24	Cure Impr.
4.	T. B.	7	M.	Eth. B., asthma (cough)		1. Strep. short c.; staph. albus. 2. Staph. albus.	12/27/23 8/ 2/24	0.1-0.5 0.1-0.3	6/10/24 6/ 3/24	Cure Cure
5.	A. C.	8	M.	Eth. B., many prot. allergies (cough)		1. Strep. long ch. 2. Strep. viridans; staph. albus.	10/ 3/23 2/11/24	0.05-0.5 0.1-0.5	1/28/24 5/12/24	Impr. Impr.
6.	G. H. C.	36	F.	Eth. L., pregnant muc. colitis		1. Staph. aureus. 2. Staph. aureus.	12/11/24 12/30/24	0.1-0.5 0.1-0.4	12/8/24 3/15/25	Cure Cure
7.	L. R.	34	M.	Eth. B., asthma		1. Strep. long ch. 2. Staph. aureus.	12/ 3/23 12/15/24	0.05-0.5 0.1-0.5	5/ 6/24 3/15/25	Cure Cure
8.	W. R. L.	71	M.	Eth. B., asthma, bronchitis		1. Staph. aureus. 2. Staph. aureus.	12/29/23 2/11/24	0.2-0.5 0.1-1.4	12/30/24 4/30/24	Impr. Cure
9.	W. F.	34	F.	Eth. R., sphen. R., max. R.		1. Staph. albus 1. Staph. albus and staph. aureus.	1/16/24 1/19/24	0.1-0.5 0.1-0.2	3/10/24 1/27/24	Cure Unch.
10.	G. P. A.	39	F.	Eth. B., asthma, mitral sten.		1. Staph. aureus. 2. Staph. aureus.	1/24/24 3/15/24	0.1-0.2 0.1-0.4	1/10/24 4/16/24	Unch. Cure
11.	F. C.	9	M.	Eth. B., chr. bronch. (cough)		1. Staph. aureus. 1. Staph. aureus and strep. short ch.	1/28/24 1/31/24	0.1-0.5 0.1-0.5	2/23/24 2/16/24	Impr. Impr.
12.	J. B. C.	65	M.	Eth. L., asthma, myocarditis, arteriosclerosis		1. Staph. aureus and strep. short ch. used at home; traveling, spoiled. 2. Duplicate.	2/23/24 7/24/24	0.1-0.4 0.1-0.3	3/ 5/24 7/30/24	Discontin. because severe reaction. same reason as - 2 -, Impr.
13.	M. E. T.	62	F.	Eth. B., sphen. B., chr. bronchit., asthma, myocarditis		3. Staph. aureus.	2/23/24 7/24/24	0.1-0.4 0.1-0.3	3/ 5/24 7/30/24	Discontin. because severe reaction. same reason as - 2 -, Impr.
14.	C. C. W.	36	F.	Eth. B., sphen. B., sphenopal. neuralg.		1. Staph. aureus.	2/ 5/24 2/ 8/24	0.1-0.5 0.1-0.5	3/13/25 3/21/24	Cure (Of asthma and cough) Impr.
15.	J. P.	8	M.	Eth. R., asthma (cough)		1. Staph. aureus.	2/ 8/24 2/11/24	0.1-0.5 0.1-0.5	5/16/24 3/24/24	Cure Cure
16.	M. McD.	28	F.	Eth. B., sphen. B., front. R., sphenopal. neuralg.		1. Staph. aureus.	1/17/24 5/ 2/24	0.1-0.5 0.2-0.4	4/23/24 6/23/24	Impr. Impr.
17.	K. C. R.	32	F.	Eth. B., sphen. B., front. R., sphenopal. neuralg.		1. Staph. aureus. 2. Duplicate.	1/17/24 5/ 2/24	0.1-0.5 0.2-0.4	4/23/24 6/23/24	Impr. Impr.

18. C. D.	11 M.	Eth. B. (severe cough and discharge)	1. Staph. albus and staph. aureus.	2/ 9/24	0.1-0.4	5/ 3/24	Cure
19. A. J. C.	34 F.	Eth. L., OMS. L., sphen. L., pregnant.	1. Staph. aureus (prophylactic against eclampsia).	2/27/24	0.1-0.4	9/10/24	Cure
20. A. W.	39 F.	Eth. B., streptococemia, chr. arthritis	1. Strep. short ch.	2/29/24	0.1-0.3	7/ 7/24	Inpr.
21. A. W. S.	34 F.	Eth. B., asthma (allergic)	2. Strep. short ch.	12/15/24	0.05-0.1	3/15/25	Inpr.
22. J. S.	39 M.	Max. L., eth. L.	1. Staph. albus.	4/22/24	0.2-0.4	10/24/24	Inpr.
23. C. McN.	28 F.	Eth. B., beg. ozena	2. Strep. viridans and staph. albus.	10/13/24	0.1-0.3	11/10/24	Unch.
24. H. C.	11 M.	Eth. B., (cough)	1. Staph. albus.	8/ 9/24	0.1-0.5	9/13/24	Inpr.
25. S. A. S.	31 F.	Eth. B. (allergic)	2. Staph. albus.	9/23/24	0.1-0.5	11/15/24	Cure
26. D. McD.	27 F.	Eth. B.	1. Strep. long ch.	9/ 2/24	0.1-0.5	11/25/24	Inpr.
27. L. H.	35 M.	Eth. B.	2. Staph. aureus.	10/11/24	0.1-0.4	12/ 8/24	Inpr.
28. E. J.	17 F.	Eth. L., mitral, regurg. (fever)	1. Staph. aureus.	1/30/25	0.1-0.4	3/15/25	Cure
29. D. P. S.	45 M.	Eth. B.	1. Strep. short c.	12/23/24	0.1-0.5	3/14/25	Cure
30. F. E.	10 M.	Eth. B., arthrit. (cough)	1. Staph. aureus.	12/ 4/24	0.1-0.5	3/ 7/25	Cure
			1. Staph. albus.	1/20/25	0.05-0.1	12/28/24	Unch.
				12/28/24	0.1-0.5	3/20/25	Inpr. (marked)
				1/16/25	0.1-0.5	3/15/25	Cure
						3/ 2/25	Cure
NONHEMOLYTIC STREPTOCOCCIC CONTROL CASES:							
1. W. W.	36 F.	Eth. B., sphen. B., sphenopal. neuralgia	1. Nonhem. strep.	9/ 3/24	0.1-0.4	12/13/24	Inpr.
2. C. H.	18 F.	Eth. B.	1. Nonhem. strep.	9/ 1/24	0.1-0.5	10/11/24	Inpr.
3. C. R. E.	30 M.	Eth. B.	1. Nonhem. strep.	9/13/24	0.1-0.5	9/30/24	Unch.
4. A. F. C.	30 F.	Eth. B., max. R.	1. Nonhem. strep.	9/18/24	0.1-0.5	2/20/25	Inpr.
5. A. M. C.	26 F.	Eth. B., front. R.	1. Nonhem. strep.	10/11/24	0.1-0.5	12/22/24	Cure
6. M. N. R.	32 F.	Eth. B., max. R.	1. Nonhem. strep.	10/15/24	0.1-0.5	12/ 1/24	Inpr.
7. D. W. R.	26 M.	Eth. B.	1. Nonhem. strep.	11/ 1/24	0.1-0.5	3/ 4/25	Unch.
8. M. L. R.	34 F.	Eth. B., sph. B.	1. Nonhem. strep.	12/28/24	0.1-0.5	3/10/25	Cure
9. F. L. R.	28 F.	Eth. B.	1. Nonhem. strep.	1/30/25	0.1-0.5	2/25/25	Unch.
10. E. E. S.	41 M.	Eth. B., asthma	1. Nonhem. strep.	1/28/25	0.1-0.5	3/13/25	Cure
TYPHOID VACCINE CONTROL CASES:							
1. E. S.	36 F.	Eth. B., sphen. B., asthma	1. Typhoid vaccine.	7/25/24	0.1-0.5	8/20/24	Inpr.
2. T. McE.	44	Eth. B., asthma	2. Typhoid vaccine.	9/29/24	0.2-0.5	12/27/24	Unch.
3. F. R.	39	Eth. B., max. L., asthma	1. Typhoid vaccine.	11/17/24	0.1-0.5	12/ 5/24	Unch.
			1. Typhoid vaccine.	11/18/24	0.1-0.3	12/ 9/24	Unch.

LXXXV.

THE PREVENTION OF CHRONIC MIDDLE EAR
SUPPURATION.*

BY GEORGE W. MACKENZIE, M. D.,
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The above was the subject assigned for this evening by Dr. Bishop when, a few weeks ago, he invited the writer to present a paper before your society. It happens to be the same as that assigned by the program committee of the L., O. and R. Section of the A. M. A. for the Detroit meeting in 1916.

The particular risk of writing a second time on the same subject is that of repetition. The average writer looks upon the repetition of his own composition as something short of plagiarizing another's; however, since the science of medicine is a growing one, it calls for revision from time to time. This, then, is the excuse for the present attempt to bring the subject up to date.

Since chronic middle ear suppuration is nothing more nor less than the continuance of the acute form, it follows that the prevention of the chronic form depends upon the curing of the acute. Granting this to be true, then we are not concerned at this time with the symptomatology, diagnosis or treatment of chronic middle ear suppuration, but only with its etiology.

The etiology of chronic middle ear suppuration ought to include every factor that has anything to do with the etiology of acute middle ear suppuration, besides every factor that has to do with keeping the suppuration going after it is once started.

If the etiologic factors of the acute condition are allowed to go unnoticed and untreated, they will continue to act as etiologic factors in keeping up the suppuration. The causes of acute middle ear suppuration are generally considered to be twofold, the predisposing and the activating. The predis-

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posing cause is some form of nasal or nasopharyngeal obstruction, of which there are several, the most outstanding being adenoids. The activating cause is one of the acute infections having a selective affinity for the upper respiratory tract, among which may be mentioned influenza, scarlet fever, measles. For the majority of cases the above answer will suffice; however, there is a minority to be reckoned with in which the above etiology will not suffice.

As to the average case, the activating cause or infection, so far as virulency is concerned, is a more or less transient factor, since it spends itself after a while, or what is equivalent to the same thing, the resistance of the body tissues manifests itself by generating antibodies, which at first weaken the infecting organisms and subsequently destroy them. For this reason the average run of cases of acute middle ear suppuration clears up spontaneously after a more or less definite duration, ten days to three weeks, depending upon the nature and virulency of the infecting microorganism and the resistance of the individual, as indicated by the amount of antibodies (active immunity) he is able to generate in a given time.

In the meantime the predisposing factor has not changed materially. Should the patient start with a second infection of the upper respiratory tract of a different kind bacteriologically from the previous one, it is possible for a second attack of acute middle ear suppuration to start all over again. Thus in both cases the predisposing factor may be the same; for instance, adenoids, but the activating factors—infections—can be different. If the patient happens to be fairly vigorous and previously healthy, spontaneous healing is very likely to follow after a normal length of time (less than three weeks), without any untoward after effects and with fairly good hearing. So long, however, as the patient retains the nasal or nasopharyngeal obstruction that predisposed to his first and subsequent attacks of acute middle ear suppuration, there remains the same probability of recurrences from every fresh attack of infection involving the upper air passages. Besides, with each attack of acute middle ear suppuration the possibility of the suppuration becoming chronic increases. The logical method of preventing recurring attacks of acute middle ear suppuration is the early elimination of the predisposing factors. The

writer recalls a case in which there was a subacute attack of middle ear suppuration in the presence of a deflected nasal septum. The family doctor who referred the case was opposed to operations. The writer, in his efforts to please the doctor, ignored the septal deflection. The patient was treated locally by every known method that could be thought of; while the family doctor was allowed to give the patient medicine internally. After several weeks of futile treatment the patient fell into the hands of a former student of the writer, who corrected the septal deflection forthwith, following which the ear discharge promptly ceased. Had the writer practiced what he had so fervently taught instead of attempting to cater to the family physician, the patient would have been spared the loss of time and inconvenience necessitated by the less radical procedure.

We are concerned tonight chiefly with those factors which tend to make an acute middle ear suppuration a chronic one. One of these factors has already been referred to—nasal and nasopharyngeal obstruction. The obstruction may take on one of several forms, the more common being adenoids and deflections of the nasal septum, less frequently nasal polypi and hyperplasias of the turbinates. It is possible for accessory sinus disease to predispose to middle ear trouble, but it is not very common except when combined with polyps which produce obstructed nasal respiration.

Besides those physical factors referred to above, there are other physical factors, found elsewhere, capable of acting as predisposing factors to chronicity; for instance, adhesive bands found in the tympanic cavity remaining after an attack of acute exudative catarrh. During an attack of acute exudative otitis media there is generally a fair amount of seromucous secretion in the middle ear spaces. It occasionally happens that the swelling of the mucous membrane of the eustachian tube continues for a comparatively long time, so that the patulousness of the tube is impaired, thus preventing complete drainage of the sticky seromucous secretion from the middle ear. Some of the more solid elements of this secretion become organized, forming so-called adhesive bands, which act as physical barriers to the drainage of purulent secretion at some future time should there develop a middle ear suppuration. When the attic region is shut off from the mesotympanum by bands

of this kind, then the acute form of suppuration does not clear up promptly, but tends rather to become chronic and more or less limited to the attic. In this case the head of the hammer and body of the anvil, especially the latter, become involved in the destructive process. This is not a rare type of chronic middle ear suppuration and is recognized in the milder grades by a perforation of the membrana flaccida with intermittent discharge. In the more severe cases polyps may be seen presenting through the perforation; in still more severe cases the polyps are observed to be of the red granulation form in contradistinction to the pale fibrous ones. The presence of granulation polyps usually indicates bone involvement.

The same exudative catarrh that produced the previously mentioned gluey like seromucous discharge, which is prone to become organized in the middle ear spaces and form adhesive bands, shutting off more or less completely the epitympanum from the mesotympanum, can also form bands between the short process of the anvil and the surrounding walls of the aditus ad antrum, and thereby act as a barrier to the drainage of purulent secretion from the mastoid antrum. In this latter case the acute middle ear suppuration is prone to take one of two courses. Either there develops, because of imperfect drainage, a severe acute mastoid empyema, requiring an early operation to effect drainage posteriorly, or else there develops a milder grade of mastoid involvement, which is usually not recognized clinically but allowed to go on to chronicity, in which case there is a chronic middle ear suppuration which is not permitted to get well, because the middle ear cavity is constantly fed with infectious material from the mastoid cells. All chronic cases of middle ear suppuration show sclerotic mastoids, due to a condensing osteitis, which can occur only in the presence of chronic mastoid inflammation. It is because of this fact that Mr. Heath of London advocates his conservative radical operation on the mastoid in cases of chronic middle ear suppuration. More recently, Barany of Upsala, Sweden, has modified the Heath operation; but in principle it is essentially the same. While it is true that every chronic middle ear suppuration presents the evidences of chronic mastoiditis, not every case of chronic middle ear suppuration presents mastoiditis and nothing more. Some present an attic suppuration

with dead bone referred to above. In the latter case the writer still feels that the modified mastoid operation, which leaves the dead bone in the attic untouched, is not good surgery, Heath and Barany notwithstanding. Furthermore, the writer does not see how it is possible to effect a cure of chronic middle ear suppuration in the presence of a cholesteatoma without removing the growth. Whenever there is a well defined pathologic process of pyogenic origin the logical form of treatment is to go directly to it and not indirectly at it, leaving the dead bone or the cholesteatomatous mass untouched. Anyone familiar with the work of Rosenow must be impressed with the fact that bacteria increase in virulency the less perfect the drainage (oxygenation), and vice versa.

Thus far we have considered the physical causes which act to impair drainage (ventilation) of the eustachian tube, the middle ear cavity, the attic, the mastoid antrum, and the mastoid spaces. Earlier in the paper adenoids were referred to as an important predisposing cause not only of acute middle ear suppuration, but also of the recurring and chronic forms. This brings to our attention the subject of so-called pyorrhea of the eustachian tube, which the older authorities stressed considerably. They felt that most cases of chronic middle ear suppuration were kept going because the infection was being fed to the middle ear cavity by a chronically suppurating eustachian tube. This prompted them, when operating on the mastoid radically, to attempt, as the final step in the operation, the closure of the eustachian tube. Galvanocautery has been used for the purpose, but more recently curettage and filing the tube have been practiced. The failures to obliterate the eustachian tube far outnumber the successes. When one examines the osseous eustachian tube the reason for the frequent failures becomes quite evident, since the osseous lumen of the tube does not present a smooth surface. On the contrary, it appears under the microscope to be quite rough and full of tiny recesses in which bacteria can lurk and reinfect the parts. More recently Yankauer has contended that a patulous eustachian tube in the presence of a perforated tympanic membrane favors reinfection of the middle ear cavity, and if either of these conditions is removed, in the large majority of cases the chronic suppuration clears up. He, therefore, designed in-

struments for obliterating the eustachian tube. After cocainizing the tube he takes a mushroom headed knife and cuts the mucous membrane in the tube from the underlying bone and then everts the membranous lining as one would invaginate the finger of a glove. The operation, like other operations for the obliteration of the tube, is more often a failure than a success; however, when it is successful, it is usually decidedly so, since the discharge ceases altogether. Whether the failures are due to retention pockets in the bony eustachian tube, which seems quite probable, or to faulty technic, or to other unfavorable factors, the writer is unable to say.

Lessened resistance to a particular infecting organism has long since been considered a factor in chronicity. Resistance and immunity have received considerable attention since Wright's memorable work with opsonins. However, there have been times when it was rather overworked, witness the testimonials in the red book, put out by the pharmaceutical house of G. H. Sherman, M. D.

The earlier claims made for vaccines in the treatment of middle ear suppuration were indeed exalted. There will be a positive place found for vaccines in the treatment of suppuration when we shall have learned more about their action, the size of the dose best suited and the proper interval between doses. The trouble with most of us humans is that when we happen to make a good hit with any particular form of treatment, we try our hardest to make that form of treatment fit every other case that seems to resemble in the least way the case in which the results were good. Then when we meet with failures, as we must in the majority of cases, we go to the other extreme and condemn the treatment altogether.

The ideal form of treatment is that which is directed toward the etiology. If the cause of the chronicity is faulty drainage, the problem is a mechanical one, pure and simple, in which we must find out in what manner the drainage is interfered with and then correct it. If it is one of lessened resistance, pure and simple, then we may discard the mechanical form of treatment and direct our attention toward raising the body resistance. If both factors are present, then the treatment must include both. Though these two factors are important in keeping up a middle ear discharge, they are not the only pos-

sible factors to be considered. The so-called dyscrasias are less frequent factors, but none the less important. The writer is reminded at this juncture of a very instructive case he saw with his associate, Dr. W. G. Shemeley. The patient was a boy who was quite sick with what appeared to be a thrombophlebitis complicating a mastoid empyema. When operation of the sigmoid sinus was about decided upon, it suddenly occurred to the writer to have a Wassermann test made, and accordingly it was advised, with the result that it was found to be plus two. Mercurial inunctions caused a complete and rapid disappearance of all symptoms, without the necessity of an operation. Had the patient been operated on and no antiluetic treatment administered, the result might have been fatal because of misapplied treatment.

Syphilis, congenital or acquired, is not the only form of dyscrasia to be reckoned with. Tuberculosis and diabetes are just as important. Recently the treatment of diabetes has been generally more satisfactory than formerly, likewise the treatment of tuberculosis. Incidentally, middle ear suppuration in the presence of either of these conditions demands consultation with a competent internist.

The number of cases of tuberculous middle ear involvement is much smaller than formerly. Besides, the number of cases of pyogenic infection of the middle ear in tuberculous patients is less than formerly. In both classes of cases heliotherapy or some other form of light treatment promises the best results and not surgery alone. To appreciate the remarkable results possible with heliotherapy in the treatment of tuberculosis one needs but visit von Pirquet's Clinic in Vienna and witness the many recoveries from tuberculosis, even in the miliary form.

Another important factor frequently overlooked in the treatment of suppurative processes generally and of the middle ear particularly is that of diet. Earlier writers did, as a matter of course, make some reference to diet as a factor to be considered in the treatment of disease, particularly in that class of cases where there was a suspicion of the so-called tuberculous diathesis. The subject of diet is beginning to be considered scientifically, especially since the International Congress of Hygiene in Washington, 1912, when the vitamin deficiency

diseases received so much attention. It has long been known that butter fat and cod liver oil act beneficially in the treatment of tuberculosis. More recently it has been found that these fats work just as beneficially in the ordinary pyogenic infections. L. W. Dean, of Iowa City, has made some very important observations along this line. Quoting from a paper by him which appears in the proceedings of the American Academy of Ophthalmology and Otology for 1923, page 269, "The second most important thing in the treatment of paranasal sinus disease in infants and young children is diet. Dr. Amy Daniels, the Research Dietician in the Department of Pediatrics in the University of Iowa, has expressed her opinion regarding this matter as follows: "We have observed that animals fed diets quite lacking, or very low, in the so-called fat soluble vitamine ('Vitamine A') are subject to paranasal sinus infections, infections of the middle ear, a condition which precedes the eye manifestations—xerophthalmia—characteristic of animals fed diets low in this vitamine. In such animals, the addition of substances, butter fat, cod liver oil, leafy vegetables, etc., which are rich in vitamine A, corrects the condition if dietary therapeutics are inaugurated in time."

The matter of a complete physical examination of the patient in order to determine the presence or absence of tuberculosis locally or more distantly requires the cooperation of the internist, roentgenologist and the pathologist, and, we might add, in some instances also the endocrinologist. If the roentgenologist happens to be a diagnostician pure and simple and does not use the roentgen rays or other forms of light for treatment purposes it is necessary to find someone else who does.

It is very satisfying to be able to call to one's assistance capable specialists in their line to share the responsibility when needed and, too, the credit in the case of success. By less complete methods many of the irregular or atypical cases do poorly and eventually slip away, to fall into the hands of the more painstaking, broader visioned experts. In the presence of diabetes or other dyscrasias the same careful cooperation of experts is required in the treatment of those cases of acute middle ear suppuration which do not promise to heal after a reasonable length of time following the elimination of all the con-

tributing physical factors, including an operation for the drainage of the mastoid spaces where mastoid empyema is found to be present.

Fortunately for the laity, the large majority of acute middle ear suppurations get well spontaneously without treatment. An even larger number get well under routine treatment. Whereas every case ought to clear up under precise treatment. The precise treatment depends upon finding out what the exact causes are in each case and removing them. This the writer has attempted to outline in his present effort. To cover every phase, and to do it thoroughly, would require much more time than a single evening and the assistance of the several experts referred to above.

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LXXXVI.

INTRANASAL AND EXTERNAL METHODS OF
OPERATING ON THE ETHMOID CELLS—A
CRITICAL CONSIDERATION.

BY ROSS HALL SKILLERN, M. D.,

PHILADELPHIA.

In presenting this subject, it is not my intention to show partiality to one method over another, but rather to assume the standpoint of the critic and by comparison of the different phases, different conditions and different results, endeavor to establish certain definite and trustworthy facts that will serve to guide us whenever it is brought up to us to make such a decision.

In considering a primary operation on the ethmoids, I do not believe any of us would have the temerity to choose deliberately the external route without having grave reason for so doing. In debating this subject the following conditions would have marked influence on our decision: (1) when repeated intranasal procedures had failed to bring about an amelioration of the symptoms and the patient demanded relief; (2) rupture externally with formation of a fistula, and (3), threatened or actual orbital or cerebral complications.

The last two postulates can, to all intents and purposes, be excluded, as the frontals are practically always coaffected and the indication is for some form of external operation on the frontal, including the ethmoid rather than on the ethmoid alone. This, then, would give us but a single absolute indication for an external operation on the ethmoid and that, when we have operated through the nose, repeatedly if you will, and failed in our purposes. Let us suppose, then, that we are confronted with such a condition of affairs. What assurance can we give the patient that such an operative intervention (external route) will bring about a cure? Let us for a moment

examine the situation. It presents two phases, (1) from the standpoint of the operator (difficulties of the treatment), and (2) from the standpoint of the patient (danger of uncertain results).

The standpoint of the operator is usually one of doubt and indecision, particularly if he has been the one who performed the previous intranasal operations that led up to the present condition of the patient. This condition consists of a large mass or masses of suppurating tissue, occupying the site of the ethmoid capsule on that side. The thought will arise, disagreeable as it may be, that there was once a time when the infection occupied a limited portion of the ethmoid (one or two cells) and, through injudicious operative work, the infection has spread until no one knows how great the involved area actually is. One may here interpose, "but the roentgen ray will give us this information." In answer, I can only say that too many bitter experiences have long since made me hesitate to accept without reserve the roentgenologists' interpretation of the lights and shadows found on the roentgen ray ethmoid film. This has gradually been brought about by the wide divergence that manifested itself at the time of operation, between the anticipated pathologic conditions and the actual operative findings.

Now, then, speaking of widespread infection (diffuse purulent ethmoiditis), I doubt whether I have ever seen a chronic case in which the entire ethmoid was purulently involved primarily, except in that atrophic type which so closely resembles true ozena, and in these cases any form of operative intervention seems to be contraindicated, as it only aggravates the infective process.

It might be a matter of interest, in passing, to state that in every fatality following frontal sinus operation that has occurred in my practice, atrophic processes were present in the ethmoid region.

Reverting, then, to the state of the patient, coupled with the indecision of the surgeon, the result will be an impasse which will be broken only by a renewed effort along other lines by the attending rhinologist, or a wandering away of the patient.

Most men do not like to perform the external operation. The idea of such extensive bone resection causes one to approach it with a certain degree of hesitation. The comparative mildness of the symptoms does not seem to warrant an operation of such magnitude, nor is the patient who has had extensive ethmoid surgery ever the same, speaking from the physiologic standpoint.

Now let us for a moment consider the actual difficulties encountered in the two operations.

THE INTRANASAL OPERATION.

The narrow nose is the *bête noire* of the ethmoid operator, particularly that type of nose that is not dependent on a deviated septum but is congenitally narrow. When such a patient acquires a general purulent chronic ethmoiditis, I do not know a greater indication for the external procedure, for any intranasal intervention will fail, owing to the inaccessibility of the remote portions of the ethmoid under such anatomic disadvantages—the nose that has suffered repeated operative interventions until the ethmoid region presents a disorganized mass of bony, fibrous and mucoid tissues, with all landmarks obliterated.

How many of us present have been called on under these circumstances? (Quite a number, if one may judge from the expressions of most of you.)

The difficulty in operating is manifold: Not only is orientation most difficult and often well nigh impossible, but the proliferation of aberrant osseous tissue following repeated operations makes complete exenteration of mass through the nose problematic, to say the least; with this condition obtaining, again the external procedure offers our only hope.

THE EXTERNAL OPERATION.

Before enumerating the difficulties, let us remember that this method is considered the ultraradical. I mean, the one in which the entire labyrinth from the lacrimal to the pre-sphenoidal cell is supposed to be exenterated, and not merely a simple opening through the rim of the orbit with indis-

criminate, but cautious, removal of parts of immediately presenting anterior cells.

As far as the difficulties are concerned, I know of no greater difference than this operation practiced on the cadaver and performed on the living. In the first instance, devoid of blood, it is a beautiful, easy and most satisfactory procedure. On the living patient, with the operative field an almost constant well of blood and with the orientation uncertain, particularly if the middle turbinate has been removed, which is almost always the case, the operation is difficult, unsatisfactory and unattractive. The experience of the operator (at least personally speaking) is something as follows:

The external incision is made, and, as soon as the periosteum is reached, a profuse hemorrhage occurs which requires considerable time and trouble to control. The soft parts are retracted, and through this narrow wound the chisel is used to open the ethmoid through the lacrimal fossa, and more bleeding from the depths of the ethmoid, which continues during the entire operation, makes it difficult to keep the field free. The cells are now attacked and removed with suitable forceps, with an endeavor to keep close to the orbital plate and as high as possible, but away from the cribriform plate with its foramina. The exenteration is continued through a greater or lesser pool of blood, until the anterior sphenoid wall is reached. The débris is now removed together with any spicules of bone, the wound is closed, and a strip of gauze is inserted for drainage. The operator is finished, but he is by no means sure that all the cells have been adequately opened, particularly those lying in the anterosuperior external angle or where the so-called orbital ethmoid cells are always situated. After the gauze is removed, on the third or fourth day, and the wound allowed to close entirely, the after treatment is precisely the same as if the internal method had been followed. Now what is the difference in the results of the two procedures, presupposing that neither of the absolute indications for the external operation had prevailed? None, if I may be permitted to answer the question, at least in my hands, for the internal route has given me quite as much satisfaction as the external, with certainly less of the trouble and anxiety that is connected with the latter.

SUMMARY.

It seems to me that the external method as a primary operation has its place only with these rare indications:

1. Pointing, or actual rupture externally.
2. In the event of orbital or cerebral complications.

As a secondary procedure, the indications are much more elastic, suited to the wishes of the individual operator; but let me say this: Should it be decided to perform the external operation, let it be done not by a mere opening and the haphazard removal of a few cells that lie within easy grasp of our forceps, but approached with the idea of exenterating the entire capsule from the foremost infundibular cell to the sphenoid area and from the fovea ethmoidalis to the lamina papyracea; for unless this is done we shall subsequently find ourselves in the same predicament as if we had done an incomplete internal operation, except that in addition we shall have burned our bridges behind us.

LXXXVII.

THE DIAGNOSIS OF LATERAL SINUS
THROMBOSIS.

BY CURTIS C. EVES, M. D.,

PHILADELPHIA.

It is a well recognized fact that an early diagnosis in infection of the lateral sinus is most important; an early diligent study of the whole complex surrounding each individual case of mastoiditis is necessary.

My personal observations are limited to thirty-eight operated cases for sinus infections, with six deaths, and two cases of metastatic abscess of the hip joints. To try to classify these cases under a few separate headings is an impossible task. In fifteen, the diagnosis was delayed until septic pictures were presented. Four were acute fulminating infections, presenting a positive diagnostic picture within seven days of the onset of the acute purulent otitis media. Two were latent cases with low grade infections, one of which showed only pain, and the other pain with mild temperature.

SYMPTOMATOLOGY.

The early appearance of the patient is usually deceptive. He looks well and feels well; later he will invariably show the anxiety and depression accompanying septic infection.

Dryness of the lips and coated tongue are important early symptoms often overlooked.

Pain of slight or severe nature over the side of the head affected, often localized in the region just back of the mastoid and reflected to the occiput and to the top of the skull, is a very suggestive symptom when other intracranial complications are ruled out. The sensation may be described as fullness, as pressure, or it may be a severe deep pounding or a steady ache. Pain in the neck, caused by the movement of the head, when the glands at the angle of the jaw and along the jugular vein are infected, is frequently present.

The patient may complain of periodic feelings of chilliness without having a chill. This is an important symptom to elicit by early questioning the patient. Inability to sleep well, together with loss of appetite and nausea, are other symptoms sometimes complained of. In some cases, pain over the sinus may be elicited by percussing the area with the finger.

Edema over the posterior portions of the mastoid drained by the emissary vein is seen as an early symptom in many cases. It is described by Tobey¹ as the most constant reliable symptom.

Tenderness of the glands at the angle of the jaw may be elicited in most acute mastoid cases, especially in children, and has no special value as a symptom of sinus infection.

Tenderness of the glands along the jugular vein occurred in one of my late cases. It is a rare but suggestive symptom of venous infection.

Eyegrounds.—In about 10 per cent of the cases changes in the form of optic neuritis are seen by frequent examinations of the eyegrounds. This sign is of importance only when other causes of intracranial pressure are eliminated.

Increased dilatation of the veins of the fundus, with increased prominence of the veins of the forehead, temple and eyelids, by deep pressure upon the jugular vein of the opposite side, as described by Crow,² is considered of value when the sinus is completely occluded. This sign has not proven of value to me, perhaps because of the faulty technic. An overdistended external jugular vein of the affected side has been a more consistent suggestive sign.

Chill.—A well marked chill has been seen in about 10 per cent of my cases. Both Dench³ and Tobey⁴ have found true chills present in less than 50 per cent of their cases. Milligan⁵ considers a rigor in cases of otitis media, otherwise unaccounted for, a suspicious symptom.

True chill is a sign that the infection has passed beyond the stage of safety. Its absence should not deter the early examination of the sinus in the presence of other findings.

Temperature.—From a normal temperature to any variety of fever may be seen. The most common temperature occur-

ring in my cases was a moderately high (102), slightly irregular one, for from two to four days, then becoming markedly irregular, with sudden rises to 103 to 105, or even higher in children, with equally abrupt drops to normal or below, occurring once in 24 hours. The sudden rises and falls may occur once every few hours (three to four), or as infrequently as once in a few days (four days to one week). A record of the temperature should be made at least every two hours. A high temperature following acute mastoiditis, not accounted for by other causes, is a very suggestive finding.

A low grade temperature, from 99 to 100 in the morning to 101 in the evening, is often seen in early stages of a sinus infection.

In the acute fulminating cases, the temperature is usually highly irregular from the start.

No temperature was observed over a period of four weeks in one.

Bacteriology.—In common with almost all other cases recently reported, I have found the infecting organism to have been streptococcus hemolyticus, in a majority of instances. Smith⁶ has found pneumococcus almost as frequently. One should not overlook the fact, Dench⁷ advises, of the insidious destructive action of the bacillus mucosus capsulatus.

Blood Count.—A daily blood count is of importance to indicate by the leucocytes the state of resistance of the patient. An increasing percentage of polymorphonuclear cells, especially with a decreasing leucocytosis, should hasten the exploration of the sinus.

The average leucocytosis is reported by many authors as between 16,000 and 20,000. In one of my cases it reached 32,000, with 90 per cent polymorphonuclear, the fifth day after the onset of O. M. P. A. In two of them it was as low as 6,000. The average leucocyte count was 17,500, with polymorphonuclear count ranging from 70 to 90 per cent.

Blood Culture.—In twenty of the cases in which record is made of the blood culture, five had bacteremia. Three of these cases were seen late and were operated upon before the report was made.

Even in some of the most decided positive cases, bacteremia is not demonstrable. One should not hesitate to operate in its absence with other suggestive symptoms present.

The presence of bacteremia in sinus infection has been found by some authors in as high as 50 per cent of their cases.

After other conditions causing bacteremia, such as erysipelas, pneumonia, acute endocarditis, tonsillitis, etc., are excluded, a positive blood finding makes the diagnosis sure.

At the Time of Operation.—The anatomy of the mastoid bone gives little or no assistance. Theoretically, a far forward sinus deeply buried in the mastoid bone is more prone to infection. Practically this has not been borne out by most observers.

In the presence of perisinus abscess or necrosis of the sinus plate, one has valuable aid for the future, should symptoms develop.

It has been my experience that a clot is more likely to be present in a sinus presenting an uneven thickened wall with loss of blue color, with yellow, gray or red discolorations, than when the sinus is protected by a layer of granulations. Sloughing or ulcerations upon the wall of the veins are pretty sure signs of an internal infectious clot.

Absence of pulsations, with a doughy or hard feeling upon palpation, indicates a clot. Pulsations may still be present if the clot is only partial. Twice I have observed, in cases having small clots with pulsations still present, a distinct thrill, similar to the sensations received when an aneurism of the aorta is present by pressing the finger into the suprasternal notch, was felt by pressing the finger upon the exposed sinus.

In the hemorrhagic type of mastoid an infection of the sinus may take place through the vessels without any visible indications of the disease of the sinus plate.

Double Mastoid.—In double mastoid a careful study of the wounds will usually reveal that one side is more diseased than the other. This is more likely to occur on the side where the infection has been of the longest duration. Both sinuses should be thoroughly exposed, from the knee to the bulb, and carefully inspected. If not sufficient evidence can be ascertained, while observing and palpating the exposed sinuses, a needle puncture may be made; if the blood comes up into the

needle more readily on the one side than on the other, especially if no blood can be aspirated from one side, it is indicative of a clot in the sinus. A culture should be taken from the blood on both sides for future study in case a diagnosis cannot be reached.

The needle puncture is not without danger and is not advised except as an aid in the doubtful cases of double mastoiditis.

The presence of the streptococcus hemolyticus, bacillus mucosus capsulatus, or pneumococcus, in the original mastoid wound of the one side, with their absence in the other, makes the differential diagnosis more certain. The right sinus is more frequently involved than the left.

In children, owing to their susceptibility to intercurrent infections, and to the sensitiveness of their temperature reaction, we are concerned with a more difficult problem. Here, probably more assuredly than in the adult, must the diagnosis be one of elimination. The assistance of a competent pediatrician cannot be dispensed with.

X-Ray.—A well made roentgen ray film usually clearly outlines the course of the lateral sinus. None of the more experienced roentgenologists claims that it is possible to diagnose a clot.

Phillips⁸ considers high temperature continuing several days after mastoid operation, especially when operative findings disclose necrosis of sinus wall and bacteremia with no other body complications, sufficient evidence to call for an examination of the interior of the sinus.

Dench⁹ says: "In a case of mastoiditis with numerous excursions of temperature extending over a number of days, it is perfectly justifiable to make a diagnosis of sinus thrombosis."

Atkin:¹⁰ "The combination of elevation of temperature, particularly of the remittent and intermittent type, chills and positive blood culture, make the diagnosis certain."

Milligan:¹¹ "To await the development of a full array of classical symptoms and signs is merely to court disaster."

Frazier:¹² "In all cases of middle ear suppurations, associated with evening fever, the sinus should be exposed and examined."

CONCLUSIONS.

1. In both acute and purulent otitis media the symptoms, signs and laboratory findings of outside complications very closely resemble lateral sinus infection; a careful physical examination should always be made.

2. The otologist, with the assistance of the laboratory findings, should be able to differentiate sinus infection from brain abscess and meningitis, the latter usually showing their characteristic group of symptoms. If doubt exists as to the differentiation an early exposure of the sinus should be made.

3. Early symptoms indicating sinus infection complicating mastoiditis are: Elevation of temperature, pain or ache over the posterolateral side of the head affected, edema and tenderness over the area drained by the mastoid emissary vein, dryness of the lips, coated tongue, loss of appetite, inability to sleep well, with otherwise a feeling of well being by the patient.

4. Provided other bodily conditions are eliminated, elevation of temperature, associated with one or more of the above signs occurring for several days after mastoidectomy, are sufficient to put the otologist on the watch for sinus thrombosis and calls for opening the sinus, should there have been evidence at the time of the mastoid operation to suspect infection.

5. In many cases the signs and symptoms are not sufficient to make a diagnosis before pyemic symptoms develop. Some of the obscure sinus infections can be diagnosed by a more careful study and intelligent interpretation of the early symptoms, signs and laboratory findings of mastoiditis.

6. To give the patient his best chance for recovery, or recovery without prolonged convalescence with metastasis, the interior of the sinus should be inspected before intermittent septic temperature, chills and positive blood culture are present.

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LXXXVIII.

THE DIAGNOSIS AND TREATMENT OF ATOPIC
CORYZA (PERENNIAL HAY FEVER).*

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The term atopic coryza is applied to the train of symptoms due to a general hypersensitive state, manifested clinically in the mucous membrane of the upper respiratory tract and of the eye. These clinical manifestations are congestion, lacrimation and itching of the eye and sneezing, nasal obstruction and discharge, symptoms at once recognized as those of "hay fever." If the condition be definitely seasonal in character and caused by pollens, it is, indeed, hay fever; but if the complaint be irregular in occurrence, the attacks bearing no relation to the month or season of the year, and due to inhaled substances other than the pollens, it should be termed a "perennial hay fever," or better, "atopic coryza."† Hence it is seen that the distinction made between hay fever and atopic coryza is not based upon dissimilarity either in the clinical manifestations or in the degree of severity of the symptoms, but solely upon the difference in the causative agents. Coakley,¹ in his book "Diseases of the Nose and Throat," gives a classification of hay fever in which he divides his cases into a seasonal or pollen group and a perennial or nonpollen group.

Since the hay fever patient almost invariably protests that he can foretell the month, day and hour when his symptoms will appear, it is usually possible, by means of a careful clinical history, to distinguish between the pollen type and the non-seasonal, or perennial, type of coryza. Not always, however,

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†The term "atopy" was coined by Coca and Cooke to denote the inherited form of human hypersensitiveness.

do patients whose symptoms are confined to a definite season of the year prove to be pollen cases. A housewife describes her coryza as occurring only within a certain period of the spring when the trees are pollinating; but on testing she proves to be negative to the pollens and very sensitive to housedust, finally admitting that the period of her sneezing and coryza coincides with the time of her spring housecleaning. Or a lad describing his hay fever as occurring each year in the month of June, when he is on his vacation, may prove to be a horse sensitive case, spending his holidays on horseback.

On the other hand, there are certain cases of true hay fever which appear to be nonseasonal, the seasonal limits being obscured by a secondary infection or by a multiple sensitization in which not only pollens but other factors as well are found to be operative.

The clinical history is of value also in determining whether the complaint is a true hypersensitive condition or a rhinorrhea or infective type of coryza, since it has been found that a positive family history occurs in 60 per cent of clinically hypersensitive cases, whereas it is present in only 7 per cent of the general population. Hence the presence of hay fever, nonseasonal coryza or bronchial asthma in the parents, grandparents, uncles or aunts, always suggests the probability of a hypersensitive reaction in the patient. Further interesting facts are obtained by combining the results of a study by Cooke and Van der Veer on the "Inheritance Factor in Human Hypersensitiveness"² with a later study on the same subject, by Cooke and the writer,³ a total of 1889 cases thus being used.

The occurrence of a hypersensitive condition in the antecedents definitely affects the age of onset of clinical symptoms in the patient. In approximately 90 per cent of cases where both paternal and maternal family histories are positive for hypersensitiveness, the onset of clinical symptoms occurs within the first ten years of the patient's life: if the antecedent family history is positive on but one side—that is, either paternal or maternal—only 30 per cent of the cases develop clinical symptoms in the first ten years. Cases with a negative family history develop symptoms, as a rule, at an even later age. Of all children with a positive bilateral family history

(that is, both paternal and maternal), over 70 per cent will eventually develop clinical symptoms; of all children with a unilateral family history (either paternal or maternal), 50 per cent will develop symptoms.

As long ago as 1872 Wyman⁴ noted that some families suffered from hay fever more than others and pointed out that family predisposition had been underrated. Beard, 1876,⁵ found that 35 per cent of a total of 190 cases had one or more relatives affected, and Mackenzie,⁶ in 1884, found a positive family history in 40 per cent of his cases.

It is possible to divide the causative factors of atopic coryza into two main groups, according to the mode of contact; first, the substances which cause symptoms by inhalation, and second, those which cause symptoms by ingestion. The first is by far the larger group, and includes all such airborne substances as dusts, animal emanations and vegetable powders, as orris, rice, corn, wheat and barley flour. The second group includes the foods and drugs.

Dusts play a very important rôle in the causation of non-seasonal or atopic coryza. There are two groups, the house dusts and the occupational dusts. The house dusts are composed chiefly of particles of lint from cotton, woolen or linen materials used in floor coverings, bedding and clothing; of glue from furniture; of talcum and toilet powders; of dander and hair from pet animals; of bits of down or feathers from upholstery or pillows. The occupational dusts, as would be expected, contain the substances characteristic of the shop or factory from which they have been obtained. Bakery dust is chiefly wheat or rye flour, jeweler's dust contains a large percentage of boxwood, furrier's dust contains many fur particles, dye substances and preservatives.

A second group of coryza cases is due to the emanations of those animals with which one comes in frequent contact, chiefly the scales and hair of the horse, dog and cat; also the feathers of the chicken, duck and goose. It is small wonder that there are so many feather sensitive cases diagnosed when it is recalled that it is a rare individual indeed who does not have his nose in contact with a feather pillow for at least six of the twenty-four hours. Since the Russian immigrants have a custom of using rabbit hair as a substitute for feathers in

mattresses and pillows, and since the Mediterranean peoples use goat hair for the same purpose, these two substances are often found to be causative agents, especially in clinic cases. Rabbit hair is also used in a variety of ways in the trades. It is employed in making felt materials and certain grades of men's hats; it is used frequently in overstuffed furniture and in the cushions of motor cars; it masquerades as beaver, coney, mink or any other fur that the craftiness or imagination of the furrier can devise. Many cases of coryza have been found to be caused by the rabbit hair used as fur trimming in coats and capes of women and children.

Face powders, sachet powders, talcum powders, tooth powders, in fact, all toilet powders, contain orris root, rice powder or cornstarch, three substances which are very common causes of coryza. Orris is considered essential to a good toilet preparation, since it is used as the vehicle to carry the perfume with which such articles are usually loaded. As would be expected, women are the chief victims. Barbers are also frequent sufferers from this form of coryza.

Cottonseed, flaxseed and glue are substances to which a number of individuals have been found to be sensitive, and usually to amounts incredibly minute. Cooke⁷ has the case record of a patient very sensitive to glue, who declared that he would develop violent coryza and asthma upon entering a room in which there was an uncovered glue pot. This statement was not verified.

Cereals are frequently responsible for cases of coryza by inhalation rather than by ingestion, especially among those who work in places where the air is heavy with the dust or flour of grain, as in the case of bakers or millers. Hops or barley are causes of coryza by inhalation among brewers and dealers in brewers' supplies. In druggists, lycopodium and powdered ipecac often cause symptoms by inhalation. Insect powders are frequent causative agents in atopic coryza.

Coryza following ingestion of foods or drugs, as has been said above, is relatively rare as compared with the inhalant group, and the cases are more difficult of diagnosis. The attacks of coryza are frequently associated with asthmatic attacks. Among the causative factors are milk, egg, cereals, fish, shellfish, nuts, mustard and chocolate. Among the drugs are

aspirin, quinin, methyl salicylate or oil of wintergreen, tincture of delphinium or larkspur.

The diagnosis of cases of atopic coryza depends upon the clinical history; the physical examination with especial attention to the nose and paranasal sinuses; and upon the cutaneous tests. These tests, which form the corner stone in the diagnosis, have been so frequently described that it is necessary here only to mention a few points which should always be borne in mind, in the interpretation of the results. First, beware of the false positive. A reaction which is apparently positive at the first testing but which cannot be subsequently verified should play no part in the diagnosis. For instance, a coryza should not be considered as due to horse epithelium if the marked positive reaction obtained at the original test cannot be duplicated at will. Second, never consider as a causative factor a substance, even though it gives repeated positive reactions, unless it can be proven that the patient comes in actual contact with the substance during the period of his coryza. Finally, it is necessary that the original clinical symptoms be artificially reproduced at will on the introduction of the substance either inhaled, ingested or subcutaneously injected.

There are a few substances which usually fail to cause positive skin reactions, even in patients who are, beyond any question of doubt, sensitive to them. In this group belong certain insect powders, made chiefly of pyrethrum, many of the foods, and certain drugs, as aspirin and quinin. We are forced here to rely on the clinical history and the ability to reproduce the condition. As an example, there may be cited the case of a middle aged woman who had severe attacks of coryza, which were known to be due to insect powder, although she was negative by cutaneous test. At one time, on returning from a short vacation, she developed a most severe attack, the cause of which could not be located until it was learned that during her absence her closed apartment, which she was careful to keep free of all insecticides, had been entered by her overzealous landlord, who sprinkled the place with roach powder.

In another case, a young woman of thirty-five, chocolate was proven to be the cause of a chronic coryza, although the skin reactions were persistently negative. Strict avoidance of choc-

olate and cocoa caused her coryza to disappear completely. When entirely free she was instructed to eat a moderate amount of chocolate, which she did. Her symptoms returned in twenty-four hours, but disappeared again under the influence of the diet. It was found that the symptoms could be repeatedly induced by this procedure. Since abstaining from chocolate she has been entirely well.

Bronchial asthma is often associated with cases of non-seasonal coryza and is due to an irritation and congestion of the mucous membrane of the bronchial tree, which is analogous to the condition of the mucous membrane of the upper respiratory tract as found in nonseasonal coryza.

Infections, nasal and paranasal, are frequently found in connection with atopic coryza and are usually chronic in nature. These cases resolve themselves into two classes. In the first class the infection, whether it be of the antrum, sphenoid or ethmoid cells, is simply coincident with the hypersensitive state and apparently bears no direct influence upon it. Its presence is only casual, and any improvement resulting from its proper treatment by a rhinologist will have only an indirect benefit, if any, upon the hypersensitiveness which is responsible for the coryza. In the second class, however, there has been found to be a more direct relationship between the hypersensitive state and the chronic sinus infection. The infection, by keeping the mucous membrane of the sinuses and nose in a congested and irritable state, greatly intensifies the susceptibility of the patient to those irritative substances to which, even under ordinary conditions, he is very sensitive. As a result of the greatly increased symptoms of coryza, the congestion and irritation of nasal and paranasal membranes are aggravated, and, through further blocking of drainage, the sinus infection grows worse. Thus a vicious cycle is established, and only through treatment of both the sinus infection and hypersensitive condition can relief be found. The most thorough and painstaking treatment by the rhinologist alone is not sufficient; the underlying hypersensitiveness must be lessened.

In the treatment of cases of atopic coryza there are two distinct measures to be considered. The first consists in removing the patient from all contact with the irritating sub-

stances, where this is possible; the second, in producing in the patient a satisfactory degree of immunity by injections of the specific irritating substance. In many cases of coryza it is possible to afford the patient complete and permanent relief by the first measure alone; this is true of many of the cases due to substances inhaled, and of almost all of those to substances ingested. On the other hand, where it is impossible to remove completely the causative substances, as in the case of dusts, orris powder, epithelia and other inhalant substances, injections must be given to afford relief.

The amount of treatment necessary varies with the individual, but as a rule improvement can be noted after a period of a few weeks in the simple, uncomplicated cases. Where the cause is obscure, or where the environment cannot be controlled, as in certain of the occupational cases, or where there is an associated infection, the results of treatment are variable and uncertain.

In conclusion, there are several facts important enough to warrant repetition. First, the distinction made between hay fever and atopic coryza is based solely upon the difference in the causative agents. Second, the tendency to atopic coryza is an inherited factor. Third, the majority of atopic cases is caused by airborne substances, comparatively few being caused by food or drugs. Fourth, a marked cutaneous reaction upon testing is of itself of little diagnostic value unless it be a constant finding and unless it be due to a substance possible of demonstration in the patient's environment. Fifth, a chronic sinusitis has frequently been found associated with atopic coryza. It is, therefore, important to consider an associated hypersensitive condition as a basic etiologic factor in certain cases of chronic sinus infection.

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LXXXIX.

THE RELATION OF TONSIL INFECTION TO
NEPHRITIS IN CHILDREN.*

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It is only in the past few years that much attention has been paid to tonsillar infection as an etiologic factor in nephritis. Few of the older textbooks on children's diseases do more than mention it, and the removal of diseased tonsils as a part of the treatment is often forgotten entirely. More recent textbooks place considerable emphasis on this, however, showing the comparative recency of this viewpoint. Current literature contains many articles devoted to this subject, but previous to 1920 it was seldom mentioned.

Physicians are not satisfied with the old conservative treatment, mainly that of diet regulation. Other methods have been sought, and it is now being recognized that in certain forms of nephritis the tonsils play an important part, not only in precipitating but in maintaining a kidney infection. This is a subject demanding a common understanding between pediatric and head specialist.

The pediatricist who does not recognize tonsillitis as an important factor in the etiology of nephritis will fail to utilize one of the most vital means of treating his cases. On the other hand, the head specialist must have some understanding of nephritis in order to know when a tonsillectomy should be employed as an aid in the treatment. Each viewing the subject from the narrow confines of his own field will fail to appreciate the relation between tonsil and kidney, and the patient is the loser.

*A Thesis presented to the Graduate Faculty of the State University of Iowa in partial fulfillment of the requirements for the degree of Master of Science, June, 1924.

A good example of the modern viewpoint is presented by Forscheimer:¹ "Tonsillar infections are another source of acute nephritis, and the child subjected to repeated attacks of tonsillitis should have the tonsils carefully removed. Such a procedure is equally important in chronic nephritis, as tonsillar and tooth infections may be important factors in acute exacerbations. Removal of tonsils lessens the danger of contracting diphtheria, which, next to scarlatina, is the acute infection most liable to be complicated by nephritis." In another place, on prophylaxis, he states: "Acute nephritis not infrequently follows very mild acute infections, especially tonsillitis. Complete removal of the tonsils is advisable in children subject to repeated tonsillar infections, not merely to avoid nephritis, but to ward off attacks of acute articular rheumatism. Gargles and sprays are probably of little value as preventive measures, as usually the infection begins deep down in the crypts, far beyond the reach of local agents. Any tonsil when associated with cervical enlargement should be removed." This is an unusually bold attitude as compared to that of the other textbooks and is more insistent than that found in many. Pfoumler and Schlusman,² however, as far back as 1905, made the following important statement: "The nephritis which follows tonsillitis is also of practical importance, because it may be readily overlooked, owing to its gradual and insidious onset. The urine contains albumin in moderate quantities, and blood, and the children feel tired and are inclined to edema. The lingering character of the trouble is often interrupted subsequently by acute exacerbations."

Although these writers did not emphasize removal of tonsils as a part of the treatment, they did recognize the tonsils as responsible for many cases of nephritis usually classified under unknown etiology. Acute contagious diseases, as a rule, were blamed in those days, and an etiology other than this was obscure. Consequently only the symptoms themselves were treated, and permanent results were the exception, unless the nephritis was only a mild complication of one of the acute exanthemata. It is for this reason that etiology has been more thoroughly studied in obscure cases, and with growing recognition of focal infection in its relation to other systemic diseases, its relation to nephritis has also been studied.

Important systemic maladies are now considered due to focal infection, and the tonsils have long been the first focus examined. Such diseases as arthritis, endocarditis, chorea and cyclic vomiting are notable examples. Just what this relationship between infective foci and systemic disease really is, no one definitely knows.

How a small focus of infection in some part of the body can bring about such marked changes in other parts, or in some cases affect the entire system, has never been definitely proved. Many theories have been advanced, but they are merely explanations of a clinically known fact. The view that bacteria are transferred through the blood stream to other points is not proved, for positive blood cultures are not obtained in such cases, and until such are found this view is not tenable. It may be held that the bacteria are spread through the lymphatics. Where there is no anatomic connection, however, this is impossible. The most logical and most widely accepted theory is that of toxic absorption. Toxins are thought to be carried by the blood stream from the infective focus to the point of injury. This makes a very plausible explanation, but no definite toxins have been isolated, and why such toxins should affect one organ and not another is not readily explained.

Of course, it may be held that the affected organ is predisposed to injury by a congenitally weak structure, but this too has not been proved pathologically. The only definite proof of a relationship between focus of infection and diseased organ is a clinical one. It is known clinically that removal of infective foci brings improvement or cure to disease in other organs.

The relation of tonsils to kidneys is also without question clinically. Hill,³ in a study of nephritis in children, states that "Tonsillitis is by far the most frequent cause of nephritis in childhood." He shows that the attack of nephritis usually follows the acute tonsillitis by several days and that it occasionally follows the removal of tonsils. He emphasizes the caution that acute symptoms should be over before a tonsillectomy is performed.

In another study of the bacteriology of the urine in acute nephritis in children, Dr. Hill⁴ is unable to prove any bacterial

cause for the disease. He reported twenty-one cases, making cultures of the urine in a sterile manner, and found sixteen cultures sterile, two containing staphylococcus albus, one containing a diphtheroid, and two containing colon bacilli. He concluded that "In the ordinary type of acute nephritis there is probably no continued growth of bacteria in the kidney during the course of the disease. It is possible that bacteria may be the cause of the condition, however, producing injury at first and then dying out. It is possible, also, that soluble toxins and not bacteria may be the cause of the disease."

The evidence is almost conclusive that this is a toxic rather than a bacterial affair. Like other infections secondary to diseased foci, the only real proof of relationship here between primary and secondary points of infection is the improvement in the nephritis resulting from removal of diseased tonsils.

In many ways nephritis in the child differs from nephritis in the adult. First, the history is quite different. In the adult the trouble is very extensive, and the history is of much importance in determining the type of disease and the prognosis of the case. This is very seldom true with the child. Often nothing in the history will give any clue as to the etiology. The symptoms first noted may be so ill defined that only by urinalysis can the diagnosis be made. Second, in the adult, purely chronic types predominate, while in the child those are relatively rare. Third, the condition of the circulatory system in the adult is of much importance; in the child it is relatively of little importance.

It is for these reasons that the usual pathologic classification, used for adult nephritis, is not practical with nephritis in children. A functional classification based on the results of various tests is of little value, both because very few of these can be accurately made and because their results do not give a sound basis for prognosis or treatment. One of the best classifications to be found was presented by Dr. Lewis Webb Hill:³

1. Acute hemorrhagic; differentiated chiefly by blood in the urine. This offers a good prognosis and is especially liable to follow acute tonsillitis.

2. Acute exudative with oliguria, characterized by edema, casts in urine, elevated blood pressure, uremia and with no tendency to become chronic. The prognosis is grave.

3. Subacute, usually following the hemorrhagic type. The patient is well, but usually shows a few red blood cells and some blood in the urine for months.

4. Chronic nephritis (ordinary type), characterized by albumin in urine and occasional exacerbations. Some severe cases resemble those in adults with much albumin, edema, high blood pressure, and a tendency to uremia.

5. Chronic nephritis with infantilism (rare). These cases are subject to headache, poor vision, albuminuric retinitis, large amounts of urine with a low specific gravity, high blood pressure, small amount of albumin in urine, and other signs of infantilism.

From this classification it will be readily seen that the chief types to be considered in this paper are the acute hemorrhagic and the subacute. The hemorrhagic variety is the one usually found following acute tonsillitis, and passes into the subacute stage when treatment is not instituted and repeated attacks of acute tonsillitis cause exacerbations of the nephritis. Some cases pass into the chronic stage of the ordinary type. During this stage, one acute exacerbation after another may follow a flare up in the primary focus, and each one is usually more severe than the preceding one. Thus more and more damage is done to the kidneys, until they are unable to carry out their function. In the chronic stage, it is doubtful whether a removal of the tonsils would be beneficial, for by this time permanent damage has been done to the kidney structure, and its reserve functional power will have been ruined. As an eliminating organ it will have been made permanently below par; so much so, in fact, that it might not stand the shock of operation. The cases to be presented are those of the acute hemorrhagic type and show typical results of the effect of tonsillectomy in such a condition.

CASE REPORTS.

M. B., a white American girl, aged ten years, was brought into the Pediatric Clinic on January 10, 1923, with the en-

trance complaint of Bright's disease, and pain under her heart. Her illness began on December 22, 1922, with a chill which caused her whole body to shake. At that time she admitted that she had a sore throat. On January 4, she complained of general malaise, the urine was bloody and there was a slight general edema.

Except for bronchopneumonia at three days of age and frequent attacks of tonsillitis, the past history was entirely negative.

The father had a mild kidney disease, one other child in the family of eleven had some kidney trouble, and the maternal grandfather died of Bright's disease. The mother had frequent attacks of tonsillitis.

Physical examination on admission showed a fairly well nourished, rather pale child of normal size, with no positive findings except for very chronically diseased tonsils and slight edema of the eyelids. Blood pressure: systolic 138, diastolic 100. Urine, reddish brown, smoky, acid reaction. Specific gravity 1016. Albumin, four plus. Blood, four plus (Meyer's test). Sugar, acetone, diacetic and indican negative. Microscopically there were 300 pus cells per cubic millimeter, many red blood cells with several cellular and granular casts. Esbach's test gave four grams of albumin per liter. The Wassermann was negative. The blood count was: hemoglobin, 50 per cent (Dare); red blood cells, 3,210,000; white blood cells, 15,400, with a differential count of polynuclears 70; lymphocytes, 29; basophiles 1.

The patient was put to bed and given a low protein alkaline diet. There was gradual improvement in her general condition and in the urine. On February 10, the urine was cloudy, straw color, slightly acid in reaction, with a specific gravity of 1003. There was a trace of albumin. Blood, one plus (Meyer's test); 300 pus cells per cubic millimeter, and a few red blood cells and no casts.

On February 11, she developed an acute attack of tonsillitis with a small paratonsillar abscess. The urine on February 12 then contained albumin two plus, blood three plus, 300 pus cells per cubic millimeter, numerous red blood cells and several cellular casts.

Following this acute exacerbation she improved gradually, and on March 10 her urine was the same as on February 10, with a blood pressure of 110 systolic and 80 diastolic.

Tonsillectomy was performed March 10, under ether anesthesia. Following this there was no change in the urine.

The patient was discharged a week after the tonsillectomy. An inquiry a year later brought the report that she was well and had had no recurrence of her nephritis.

Case 2.—O. W., a girl, twelve years of age, entered the Pediatric Clinic May 2, 1923, with a complaint of headache, pain in her back and sides, shortness of breath when lying down at night and puffiness of the face and feet. Her trouble began a week before admission, with severe frontal headaches. She then had a dull, aching pain in the middle of her back and over her kidneys. Two days before admission she had edema of the eyelids in the morning and the day previous, slight swelling of the feet. The headache became worse and at night there was much dyspnea. She felt tired and weak but not drowsy. She admitted sore throat three days before her trouble began.

The family history was negative, except that the two brothers were said to have bladder trouble.

She had had whooping cough at five years of age, diphtheria at five, measles at seven and frequent colds with much nasal discharge.

Examination gave the following positive points: Slight puffiness of the eyelids but no edema, considerable pallor, very large cryptic tonsils, with much infection of the pillars, and a large mass of adenoid tissue. The blood pressure on May 3 was, systolic 174 and diastolic 130; May 4, systolic 140 and diastolic 120. The urine was cloudy, brownish color, acid reaction, with a specific gravity of 1023, with albumin three plus and blood (Meyer's test) four plus. Sugar, acetone and diacetic, were negative, and indican four plus; 150 pus cells per cubic millimeter; numerous red blood corpuscles with several cellular and granular casts. Esbach test gave one-half gram per liter. The blood count: hemoglobin 75 per cent, red blood cells 4,500,000, white blood cells 13,800; differential count, polynuclears 63 per cent, lymphocytes 34, transitional 3. The blood Wassermann was negative. An intradermal tuberculin test of

one milligram, old tuberculin, was negative after twenty-four hours and forty-eight hours. Fundi examination was negative.

Blood pressures:

May 6.....	Systolic 142	Diastolic 110
May 8.....	Systolic 120	Diastolic 90
May 10.....	Systolic 112	Diastolic 82
May 26.....	Systolic 94	Diastolic 58

The patient was put to bed and given an alkalin low protein diet. Daily examinations of the urine were made. It gradually cleared up, and on May 24 gave the following: Clear, amber, acid reaction; specific gravity, 1020; albumin, trace; blood (Meyer's test), negative; ten pus cells per cubic millimeter, a few red blood corpuscles and no casts.

On May 24 she had her tonsils and adenoids removed and was returned immediately to the pediatric wards and her diet resumed. Daily urine examinations revealed no change in the kidneys as a result of the operation. She was discharged on June 3.

Urine examinations were made on June 3 and June 18, and only a faint trace of albumin found each time, no red blood cells, blood or casts being present. Inquiry made almost a year after the operation finds her perfectly well and with no more kidney trouble.

These cases are quite typical of nephritis caused by tonsil infection, and show the results obtained by the elimination of the primary focus. Each case presented very similar urine findings: much albumin, very much blood, numerous red blood cells and pus cells, and many cellular casts. In each, except for the persistence of a faint trace of albumin, the blood and red blood cells were the last to clear up under dietary treatment. Neither gave a history of acute tonsillitis preceding the kidney affection or associated it with the trouble except by special questioning. Each showed, on special examination, tonsils that were chronically diseased and signs of repeated acute attacks of inflammation. The first evidently proved a direct relation between tonsil infection and nephritis during her stay in the hospital, since she developed an acute attack with an immediate exacerbation of her urinary findings after the urine had become practically normal. It is surprising that

neither case showed any urinary change after the operation. Each had a very mild local reaction following the removal of the tonsils, and this, with the best of hospital care and diet regulation, probably prevented such an occurrence.

CONCLUSIONS.

The tonsils should be kept in mind as a possible source of infection in every case of acute nephritis in children. Special questioning about attacks of sore throat and cervical adenitis should be included in taking the histories. Examination of the tonsils for evidence of chronic infection should never be neglected.

Medical treatment, including dietary regulation, will not suffice for the permanent cure of nephritis. A source of infection must be sought and eliminated, and the tonsils may be the offending focus.

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XC.

THE STATUS OF THE SOCALLED MIDDLE
TURBinate.

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Should the middle turbinate be sacrificed when operating on the ethmoid labyrinth?

It may be safe to assume that the consensus of opinion among operators favors a partial or entire removal of the middle turbinate, preliminary to or during surgical assault on diseased ethmoid cells.

Professional men, like laymen and sheep, are prone to follow the leader, and so we find medical writers accepting as fact the theories propounded by their leaders, with more or less careless delight. Careless, because it is the easy way. They have not deemed it worth while to think out for themselves or to investigate, by personal application of energy, thus verifying, as far as possible, the truth or falsity of the proposition or theory under discussion, whether it be a question of anatomic relation, organic function, or what not.

Thus we find passing from writer to writer, through the years, statements and descriptions of diseases or operations which, if clearly and logically put before the reader, may, if unquestioned, become classic and authoritative.

Thought is food to him who has power and energy to use it. The right to question fact or theory, to doubt, is his. Doubt and questioning stand for progress.

With this in mind, we approach the question of removal, wholly or in part, of the middle turbinate, a procedure which apparently has become classical and standardized.

Should we be allowed to question the wisdom and truth of this teaching or not?

By all means, yes!

We must then hastily review the anatomy, physiology and mechanics, so to speak, of these structures in order to arrive at some conclusion as to their function, their relative value, their comparative value; which contributes the more to normal fossa function; which should be sacrificed; which conserved, or should both alike be removed?

The middle turbinate does not develop as an independent bone, but as an integral part of the medial plate or wall of the ethmoid capsule, and the overhang covers as with a lid, the ostia of the anterior ethmoid cells, the frontal and maxillary sinuses, as well as a number of secondary turbinates, and furrows.

The middle turbinate is relatively large and is covered by a thick ciliated mucosa, rich in blood supply, the veins forming a network which gives an erectile character to the mucosa. Many mucoserous glands furnish, primarily, protection and moisture to the mucosa. Any amount, more or less than this, if continued, is abnormal and should be considered as due to a diseased mucosa.

It takes relatively a greater or less secretory activity and an augmented or diminished blood supply to accomplish this, where the mucosa is subjected to passage of air currents more or less humid, and with a variable degree of temperature. The amount of fluid given off to the inspired air is incidental to the warming process and to its percentage of humidity.

The ultimate destiny of inhaled air is the terminal or pneumatic cell, where final gaseous interchange takes place, oxygen being given off and carbon dioxid and watery vapor taken on.

In other words, the greater part of the moisture in the exhaled air is supplied by the lungs and not by the nose. Otherwise the inspired air would be saturated and not be able to remove moisture from the lungs. It is to be doubted that in very cold weather the nasal furnace is able to heat the inspired air to body temperature by the time it reaches the larynx. Under certain conditions, as superheated external air, the expired air may be lower in temperature than the inspired air. Not only are the turbinates warming and protective tissues, but they have also another function, very important but often overlooked, that of concentrating and directing a rapidly

moving current of air over and across the various ostia of the paranasal sinuses located beneath their protecting folds, thus tending, by suction, to empty those cells which are not aided by gravity as well as those which are.

The middle turbinate is important because of its size, shape and position, and because of its important functions.

First, to protect itself, by keeping the mucosa protected and moist under all atmospheric conditions.

Second, incidentally to supply heat and moisture to the inspired air.

Third, the glands of its relatively large surface secrete a substance which, if not germicidal, is at least germ inhibitory.

Fourth, it hangs as a protecting lid over the openings of the various paranasal sinuses.

Fifth, by its shape and location it directs a current of air over the hidden ostia, thus tending to keep clean the sinuses from abnormal secretions.

Sixth, it is a surgical landmark when left intact, a serious handicap when removed.

Seventh and most important, its medial wall carries the olfactory nerves, which, when cut off, allow direct communication through their sheaths to the brain, being without doubt the path of infection that causes meningitis, following nasal operations that remove a part or all of the so-called middle turbinate. (J. Parsons Schaeffer.)

What about the ethmoid cells?

Let it be stated at once that their function is debatable, and many explanations have been advanced to account for their presence.

Let it be recalled that these cells are also lined by a ciliated mucosa, but much thinned out and lacking the abundant blood supply and richness of mucoserous glands which we find on the turbinates. The ostia are very small, and often are placed at the least dependent part of the cell, so as not to admit of free and easy gravity drainage. The interchange of air between these sinuses and the nasal fossæ must indeed be slow and precludes the possibility of their adding appreciably to the heat and moisture of the inspired air. They may possibly be vestigial remains of evolutionary olfactory sinuses now devoid of nerves (in man and ape), serving to make the bones of the

face lighter; to protect the eye from extremes of temperature by intervening a series of sinuses filled with warm air. They may, in a measure, modify voice sounds. When symptoms point toward disease of these sinuses, is it logical to sacrifice a turbinate of known functional worth, to get at and remove cells of conjectural, and at most, of little physiologic value? We have taught and practiced conservation of the turbinate in these cases for years, and so universally successful are the end results that we have come to look upon the removal of the turbinate, in whole or in part, for the sole purpose of treating or exenterating the ethmoid cells, as meddlesome and dangerous surgery, as noted by the many cases of meningitis, and should be discontinued. One of the best authors states: "I think the preliminary resection of the middle turbinate, followed by appropriate treatment and careful observation before further surgical procedure is adopted, will very largely solve our ethmoidal problem." May we be permitted to ask why the preliminary sacrifice of a turbinate of known functional value, to observe and treat cells which admittedly cannot be differentiated by inspection through the nostril? The cells are too small and the space is too narrow to designate which cells of the groups are affected. Even if it was possible, the surgery of one or more cells would so injure the remaining cells as to promote trouble.

When we have good reason to suspect ethmoid disease of surgical importance, we exenterate the anterior ethmoid group or the entire capsule, as thoroughly as possible, leaving the turbinate untouched. When finished, these cells have been transformed from several cells into a clean, single cell, into which empties the frontal and maxillary sinuses, while we have, in addition, our unmolested turbinate, standing as a landmark and contributing its little part to a smoothly functioning machine.

XCI.

THREE CASES OF OTITIC BRAIN ABSCESS.*

BY S. MACCUEEN SMITH, M. D.,

PHILADELPHIA.

It is not my purpose to enter into a discussion of the etiology, symptomatology and diagnosis of otitic brain abscess, but simply to present some interesting cases.

It has been my good fortune in the past to report cases of otitic brain abscess where in more than 50 per cent the abscess was located, drained and the patients recovered. Those to be presented to you today are all fatal, but are interesting if for no other reason than to point out our faulty methods of brain localization, as illustrated in the first case—this in spite of the fact that much progress has been made in this respect in the immediate past.

Although most brain abscess formations are still found to complicate chronic otorrhea, we must keep in mind the increasing frequency of their occurrence in connection with acute otitic disease. While the early stage is usually ushered in by well recognized symptoms, such as pain, perhaps nausea or projectile vomiting, and frequently a decrease of the otorrhea or its sudden cessation, some cases are largely atypical and consequently misleading, as they show few of these early manifestations.

A change in the patient's disposition, as, for instance, when a bright and talkative person becomes dull and morose, or vice versa, has been, in my experience, a fairly constant and valuable symptom. Lowered, even subnormal, pulse rate, temperature and respiration are regarded as diagnostic and therefore most important symptoms of pus formation within the cranial cavity, and yet a reversal of this symptomatology is at times so radical that sinus thrombosis can be reasonably suspected in one instance, or meningitis in another. A recent case gave the characteristic symptoms of the early stage of

*Read before the American Otological Society, at Washington, D. C., May 4, 1925.

intracranial pus formation, but later became more pathognomonic of sinus thrombosis. Operation revealed the sinus to be healthy, but exploration showed the pus located in the temporo-sphenoidal region.

Cerebellar Abscess.—H. G. L., aged 40 years, a civil engineer of international repute, was first examined by me on January 22, 1923, giving a history of an intermittent discharge from the left ear extending over a period of nine years. He was increasingly dizzy during the first five years of his ear disease, finally reaching the point where he would at times stagger while walking along the street. His vertigo and mental confusion were greatly exaggerated in the intervals between the acute exacerbations. He experienced so much relief when the ear discharged freely that his conception of skillful treatment was the establishment of a continuous otorrhea.

One year previous to consulting me the patient had a poly-poid growth removed from the external auditory canal, which he stated bettered his condition considerably in the way of a decreased and less fetid discharge and a cessation of the vertigo.

My examination showed the otorrhea to be rather copious, brownish yellow, and very foul smelling, indicating extensive bone involvement. The membrana tympani and ossicles were totally destroyed. The tympanic end of the posterior osseous wall had disappeared through carious erosion, exposing part of the mastoid antrum. Hearing by aerial conduction was practically nil, while the bone conduction was less than one-half normal. There was a total absence of all evidence of mastoid disease over the process.

The X-ray findings by Dr. Willis F. Manges were as follows: "The right mastoid is of fairly large cell type and normal. The left mastoid is densely sclerotic, with destruction of the roof of the meatus at its distal end, and perhaps this area of destruction extends into the antral region, giving the appearance of an active necrotic process. The lateral sinus is superficial, unusually far forward, and occupies a considerable part of the mastoid antrum."

In accordance with the clinical and X-ray findings, a radical mastoid operation was advised, and the patient readily accepted this decision, but on account of urgent business found it

necessary to postpone the operation for a few weeks. In order that he might complete his work the more rapidly, he went to his camp in New Hampshire, and one day, while skating, he became dizzy, fell and remained unconscious for several hours. He was advised to return home for operation, but his improvement was so rapid that he remained at camp, finished his work, and came home apparently as well as usual, and therefore did not further consult me.

This continued for about one year, when he became unconscious on the street and was taken into a near by hospital. On regaining consciousness he complained of a severe temporo-frontal headache of the left side, for which he was treated, without relief, for some days. The ear was not discharging at this time and therefore did not attract the attention of the surgeon. Some time later the otorrhea again appeared and a radical mastoid operation was performed.

Following the operation the patient grew progressively worse, and I was called in consultation. He presented the clinical picture of meningitis, and the laboratory findings confirmed this diagnosis, with the pneumococcus as the infecting organism. Notwithstanding this obvious meningitis, I thought that the patient was suffering from a brain abscess and that the meningitis, if it actually existed, was secondary to this abscess.

About three weeks after my examination I received word that the patient was up and walking about and would soon be discharged from the hospital.

Aside from other measures the treatment had consisted of numerous lumbar punctures and the intravenous injection of mercurochrome.

At the end of the following two weeks diplopia developed, together with some nausea and the return of the temporo-frontal headache. At this time the patient consulted me, owing to the fact that his surgeon had left the city for an indefinite period. On examination I found considerable granulation tissue, rather copious, yellow, nonoffensive pus and evidence of some additional osseous involvement, as well as a Bell's palsy.

On July 24, a secondary mastoid operation was performed, after which the patient greatly improved, the Bell's palsy

almost wholly disappearing, with complete relief from the nausea and diplopia. This improvement continued for almost two months, until September 10, when the patient suddenly presented indications of intracranial pressure, manifested by lowered pulse, respiration and temperature, together with periodic stupor, but an entire absence of localizing symptoms.

Dr. Hobart A. Hare and Dr. Francis X. Dercum were in frequent consultation, but were unable to assist in locating the obvious intracranial lesion. To quote from Dr. Hare's notes: "The point of interest was that the patient had no localizing symptoms whatever, and another peculiar point about the case was that on some days his intense apathy would clear up to such an extent as to give his family much encouragement, and then on the next day he would be as stuporous as before, even to the extent of being unable to swallow, or perhaps a better term would be unwillingness to swallow, followed again by a period of 'pick up.'"

Repeated neurologic examinations by Dr. Dercum were wholly negative, as were also the ophthalmologic examinations. To quote from Dr. Dercum's notes: "There was an entire absence of any symptoms pointing to a cerebellar involvement. It was this rather negative feature which suggested to my mind the propriety of making an exploration of the temporo-sphenoid region first. There were some diffuse motor disturbances referred to the extremities of the opposite side, which were, I believe, observed by the nurse only. Loss of power could not be demonstrated in the extremities at any time except that arising from general weakness."

Prior to this date his blood picture had been practically normal, except that the hemoglobin was somewhat reduced. Subsequently the polymorphonuclear percentage and the leucocytosis were somewhat above normal, but threw no light on the subject except as indicating a moderate septic absorption. During this latter period his hemoglobin rose from 75 to 87 per cent.

Owing to the fact that intracranial otitic abscess formations are mostly situated in the temporosphenoidal lobe, and also on account of the patient's previous head pain in the temporo-frontal region, an intracranial operation was decided upon for

exploration of the temporosphenoidal and frontal regions, with negative results. We also contemplated exploring the cerebellum at this time, but on account of the patient's weak condition it was thought advisable to postpone any further operative procedure.

The ventricle was drained, which relieved the intracranial pressure and made the patient very much more comfortable for a few days, when he became unconscious and shortly died.

We were fortunate in securing an autopsy, which showed a cerebellopontine abscess of the left side.

Autopsy report, Jefferson Hospital: Body is that of an adult male, showing a marked degree of emaciation and poor musculature. Head and body hair is normal in amount and distribution. There is no evidence of deformity. Eyes are negative. Teeth in only fair condition. Just above the left ear is a recent operative incision from which there is a small amount of drainage. The surrounding tissue is normal. Chest and abdomen are flat. There is no edema. Postmortem rigidity and lividity slight. On incision of the skin, the subcutaneous fat is practically absent.

Peritoneal cavity is free from fluid. Omentum is adherent around the appendix. No other adhesions present. Abdominal organs occupy their usual position.

Left pleural cavity is interrupted by numerous adhesions; no fluid; right pleural cavity is the same.

Pericardium is normal.

Heart (240 gms.) is rather soft and muscle is brownish in color. There is no lesion of the cavities or valves.

Lungs—Left (550 gms.): Upper lobe, apex is scarred; is nodular. Lower lobe, posteriorly, is also nodular, and on section through the upper lobe there is marked increase of fibrous tissue in the apex, and about the center of the apex is a small cavity which measures 2 cm. in diameter, with well defined wall. The surrounding lung tissue in both upper and lower lobes contains numerous small nodules. The intervening lung tissue is rather red but aerated. Right lung (530 gms.) also contains numerous nodules throughout the upper and lower lobes. Many of the nodules have small caseous areas. No cavities on this side. Peribronchial lymph nodes on both sides are enlarged, rather soft, and a few are caseous.

Spleen (180 gms.) is soft in consistency, deep red in color. On section the markings are fairly distinct.

Adrenals appear normal.

Kidneys—Left (155 gms.) is fairly soft, capsule strips readily, surface is smooth. On section the cortex is of normal thickness and markings distinct. Right kidney (130 gms.) is similar to the left, with the exception that there is a small soft gray nodule in the cortex, which measures approximately 4 mm. in diameter.

Ureters, bladder, prostate and testes appear normal.

Gastrointestinal tract shows no evidence of gross lesion.

Liver (1580 gms.) is rather soft in consistency and on section cut surface markings are indistinct and the organ is rather pale.

Gall bladder is long and pendulous; no lesion observed. Duct is patulous.

Pancreas appears normal.

Blood vessels show no evidence of gross lesion.

Brain (wt. 1330 gms.)—Scalp and calvarium are normal with the exception of an operative wound behind and above the left ear. Externally there are adhesions around the dura at the point of operation which exposed the middle and internal ear. On removing the dura, over the vertex of the brain there was no evidence of inflammation of the meninges. At the base there is a large excess of clear fluid, but the meninges are not thickened or inflamed. On exerting slight tension on the brain on removing, a large amount of thick, greenish, necrotic material escaped from the anterior portion of the left lobe of the cerebellum. Upon examination a large necrotic area was found in the left lobe of the cerebellum, anterior portion, also involving the right side of the pons. The middle and internal ears were exposed on the left side, and while the bone in the middle ear is soft and necrotic in areas no pus was found. Other sinuses and right ear appear normal.

Diagnosis: Cerebellopontine abscess, left. Chronic fibrocaseous tuberculosis, lungs, bilateral. Chronic adhesive pleuritis, bilateral. Operative wound, temporal region, left side.

Bacteriology: Stained smear from the brain abscess shows a large amount of pus and many Gram positive cocci, some in clumps. No acid fast bacilli were found. Cultures show a

growth in pure culture of staphylococcus albus. Heart's blood culture remains sterile.

Histologic diagnosis: Acute abscess of brain; bronchopneumonia; chronic fibrocaceous tuberculosis of lungs; acute military tuberculosis of lungs; passive congestion of liver.

(Signed) B. L. CRAWFORD, M. D.

This case is illustrative of the fact that abscess formations do occur in the cerebellum without producing localizing symptoms. True enough, the patient did suffer from vertigo and some defect in locomotion in the early stages of his disease and long before I saw him, but this phase had entirely disappeared. It was reasonable to assume, under the circumstances, that these former symptoms were due to his labyrinthine disturbance, especially as there were no abnormalities in his gait at this time.

Temporosphenoidal Abscess.—J. W. C., aged 43 years, was admitted to the hospital September 10, 1924. At age seven, thirty-six years ago, the patient suffered from an acute suppurative otitis media of the right ear, with spontaneous rupture and a free discharge for five weeks, and then intermittently for about one year, after which it gradually "cleared up." The patient further stated, however, that it was necessary occasionally to remove "wax" from his ear, often offensive in character, indicating that the discharge had not definitely ceased.

In November of 1919, or about thirty years after the beginning of his suppurative otitis media, the patient suffered from an attack of influenza, accompanied by a recurrence of the aural discharge, if, indeed, it had ever ceased. The otorrhea became definitely intermittent, complicating frequent "colds."

In May of 1924, the man was operated upon for hernia, and he gave a history of complete cessation of the otorrhea while he was in the hospital.

In August, 1924, nearly four years after the attack of influenza and resulting otorrhea, on account of severe pain on the right side of his head, he was compelled to remain home from work. An examination of the ear at that time showed a complete obliteration of drumhead and ossicles and no discharge. There was neither pain nor tenderness in the mastoid region

and no drooping of the posterosuperior osseous wall; no dizziness.

An ophthalmologic examination showed the eyes to be normal, as were also all of the reflexes. Twenty-four hours later the patient was suddenly seized with chills and fever, the temperature ranging between 102 and 105 degrees, with rapid pulse, and pain diffused over the right side of the head, becoming gradually more severe. Eighteen hours later a profuse discharge escaped from the right external auditory canal, which relieved the head pain, fever and symptoms generally. The patient returned to his work a few days afterward, the discharge having ceased.

Ten days later he received an injury to his head, and this was followed in twelve hours by a return of the chills and fever and marked prostration. The otorrhea reappeared, lasting for three days, and then the man returned to work, still refusing to enter the hospital.

In the early part of September he was again seized with chills and fever, severe head pain and copious nosebleed, some dizziness, temperature 101 to 104 degrees, with profuse perspiration, followed by marked prostration. The pupils reacted slowly to light and accommodation, but were equal. The ophthalmologic examination was negative. Five hours after the onset of this attack a profuse otorrhea appeared, and the patient became temporarily comatose. When he regained consciousness he suffered from severe nosebleed and was unable to take nourishment, but still refused to enter a hospital. Early the following morning he became violent and showed marked signs of meningitis, soon became unconscious and was taken to the hospital, where a tentative diagnosis of brain abscess was made.

During the mastoid exenteration the dura was found to be exposed through the tegmen antri and tegmen tympani, additional bone was removed, and an abscess in the temporo-sphenoidal lobe was evacuated through the avenue of infection. The patient's condition was markedly improved the following day, and this continued for forty-eight hours, when he became quite irrational, with respiration rapid and shallow, an increased pulse rate and a considerable rise in temperature. Pneumonia had been suspected and was now recognized as a

frank lobar type, the patient dying of this disease two days later.

The probabilities are that this patient had had a brain abscess formation for a considerable time before he came under my care, and furthermore, if such was the case, it did not seriously interfere with his work until a few weeks before he was admitted to the hospital and operated upon. No doubt the extradural abscess was present for some time, and had he not been so averse to receiving treatment and submitted to an operation some weeks, or perhaps even months, before, his power of resistance might have been sufficient to bring him through the attack of pneumonia, if, indeed, this might not have been entirely averted.

Temporosphenoïdal Abscess.—R. W., aged seven years. When $4\frac{1}{2}$ years old this patient suffered from an acute suppurative otitis media of the right side, with spontaneous rupture of the membrana tympani, and a discharge which cleared up under treatment within a reasonable time.

One year later, March of 1922, she had a severe attack of influenza, complicated by pneumonia, and multiple abscesses occurring in rapid succession—of the right arm, left wrist, left side, back, right hip and left occipital region—all of which continued to drain for a period of five months.

In December, 1922, nine months after the onset of influenza, the child developed pain in the head, constant and of increasing severity, extending over a period of several months, but not at any time referable to her ear until, in August, 1923, eight months after its onset, there was a spontaneous rupture of the right membrana tympani, which relieved the head pain. This being the first intimation to the attending physician that ear disease might be the cause of the child's head pain, he had not made an aural examination, which shows the necessity of a routine ear examination, especially where the diagnosis is doubtful.

Soon facial palsy and bulging of the right eye developed, and for two weeks prior to entering the hospital the patient suffered from projectile vomiting and could retain nothing whatever in her stomach, and in consequence was greatly emaciated when she reached the hospital, and semiconscious.

A mastoid operation was at once performed, September,

1923. The cortex was bluish gray, quite thin, the cells were broken down and the cavity, which was unusually large, was filled with free pus throughout, giving a picture of an empyemic mastoid of long duration. The sinus was largely exposed through carious erosion and showed unusual pulsation, but was not otherwise involved. The tegmen antri and tegmen tympani were intact, but showed some evidence of pathologic changes.

A surgical exposure of the dura was made. The latter was tense and transmitted a faint pulsation. A large abscess, containing fully two ounces of pus, was located in the temporal lobe and evacuated.

The following day the child returned to consciousness, was bright, retained food, and the right eye was normal in appearance. She increased in weight and finally was up and walking about the ward. After several weeks of favorable progress her restlessness returned, she showed lack of interest in her playmates and surroundings, and a marked desire for sleep, persistently lying on the affected side. She was suddenly attacked with violent vomiting, a convulsion, a rise of temperature to 106°, and death immediately followed. As soon as these unfavorable symptoms developed, my colleague, Dr. J. Clarence Keeler, who was just finishing an operation in the hospital, was notified, and within fifteen minutes was at the patient's bedside, but she had died in the meantime.

Although there were some abnormalities found in practically all of the viscera at autopsy, they have no particular bearing on the intracranial lesion and are therefore omitted.

Autopsy, Jefferson Hospital: Body is that of a poorly nourished female white child, approximately 6 years of age. The scalp is covered by a luxuriant growth of light brown hair. Behind the right ear is a wound approximately 2 cm. in diameter, the floor of which is covered by a tough yellow membrane. It extends as a sinus upward and inward towards the cranial cavity. There is no evidence of discharge in either external auditory meatus. The nose appears normal. The mucous membranes of the mouth are pale and dry. The teeth are in fair condition, though dark in color, and the edge of the gums is covered by a slimy exudate. The chest is long and narrow. Abdomen is flat. External genitalia appear normal. Extremi-

ties are rigid and wasted. There is no clubbing of the fingers or toes, though the nails are cyanotic. There are numerous scars over the body—a linear scar over the right deltoid, a long irregular one over the left ulna, near the wrist, also a depressed scar on the left iliac crest posterior to the midaxillary line. These scars are firm and anemic. A recent vaccination wound is observed over the left deltoid.

Brain weighs 1735 gms. The skull is exceedingly thin. The dura is easily removed from the calvarium. The right temporal lobe is adherent to the petrous portion of the temporal bone, and the wound described, posterior to the right ear, extends directly into the cranial cavity. The right hemisphere is flattened and soft. The basal surface of the right temporal lobe is flattened. This surface is pale gray, mottled by patches of light green. This temporal lobe has the consistency of a thick gruel. Directly in the center of the basal surface of this lobe is a gray area about 1 cm. in diameter which was adherent to the dura. This area is even softer than the surrounding tissue. The dura over the petrous portion of the right temporal bone is red and shows a small gray area corresponding to the one described on the surface of the temporal lobe which extends into the middle ear. Brain hardened for further study.

On later incision, it is found that the entire temporal lobe is necrotic. In its central portion is a well defined abscess cavity 5 by 3 by 3 cm. in size, containing some viscid pale green exudate. The wall is also pale green, shading gradually to gray at the limits of this lobe.

Diagnosis: Cerebral abscess (temporal lobe); acute meningitis; suppurative otitis media (right); mastoiditis (right); acute splenitis; fatty liver; myocardial degeneration—dilatation; chronic suppurative osteomyelitis of left ilium; congestion of the kidneys.

Histologic diagnosis: Cerebral abscess; suppurative osteomyelitis ilium and temporal bone; kidney, congestion; liver, fatty metamorphosis; blood culture taken a day before death, negative; blood culture taken at autopsy, negative; smear, pus removed at the time of operation showed many pus cells and gram positive cocci; culture of pus showed staphylococcus albus and bacillus pyocyaneus.

(Signed) FRANK W. KONZELMAN, M. D.

The tube was kept in situ until some time after all drainage ceased. The sudden death in a patient who had seemed on the road to recovery may have been due to an embolus.

These three cases impressively show the almost entire absence of neurologic and ophthalmologic symptoms that we sometimes find in cases of brain abscess involving either the temporal or cerebellar regions, which is, of course, atypical, but nevertheless, their absence must be taken into consideration when a diagnosis is being evolved.

XCII.

THE THERAPEUTIC AND PROPHYLACTIC VALUE OF TONSILLECTOMY IN THE ACTIVE STAGE OF VINCENT'S ANGINA.

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Vincent published his first report, with demonstration of the bacillus fusiformis and spirochete, in the Archives internationales de laryngologie in 1898. Much literature and discussion have followed, although, until 1913, they were more or less spasmodic. From that time until 1922 there have been no less than one hundred and fifty papers devoted to this most interesting infection. Shortly after the outbreak of the World War, in 1914, the disease became very prevalent among soldiers both in base hospitals and in the field. In the British, French and Austrian armies it was a tremendous factor in disabling men for a longer or shorter period of time. Bowman cites fifty cases in one battalion. It was very natural, therefore, that considerable attention was given to the study of this disease during the war and since the proclamation of peace.

Notwithstanding the extensive observations already made, the problem is far from solution as to bacteriology, pathology and treatment. In order to justify the treatment advocated in this paper, it is desirable to review briefly that portion of the literature which has a more or less direct bearing on this phase of the subject, particularly as no one, within the knowledge of the writer, has advocated the same therapeutic plan of treatment. There is no pretense of presenting an exhaustive study of the literature. Only a few of the articles which bear directly on the subject are quoted.

Pusatori reported a case in which the tonsils were attacked, without stomatitis, and remarkable particularly because of the

chronicity of the process, which did not heal until it had persisted about one year.

Texier (1913) also reported a grave case with typical localization in the tonsils, which was treated, he says, by all the usual methods, methylene blue, neosalvarsan, etc., without arresting the progress of the affection, which spread to the base of the tongue, epiglottis, posterior laryngeal wall, and which was followed by death.

Davis and Hequet (1920) distinguish cases of acute and chronic development, and say that in the chronic cases there is no febrile reaction, but that there is remarkable destruction of the tonsils.

Kiefer (1920) claims that fusiform bacilli and Vincent's spirilla cannot be transmitted by inoculation.

Gerber, as early as 1911, recognized the infection as a mixed one and held that Vincent-Plaut's angina is only a special clinical form in a series of infections which are based all on the same microscopic findings. He also states that all ulcerations of this type have been favorably influenced by salvarsan.

Vaumosuenk (1920), on the other hand, suggested that in more than one-half the cases of stomatitis, Vincent's angina plays a secondary rôle.

Pilot and Braun (1923), in a study of the organism from the extirpated tonsils of children, found fusiform bacilli in 82 of 100 cases, and spirochetæ in 25 per cent of the tonsils, and remarked that tonsils and adenoids may be important sources of infection from these organisms and associated bacteria. In this work, the organisms were cultured in all cases.

Davis (1923), in a comprehensive study, found that the tonsillar crypts frequently harbor bacilli, which are always associated in symbiosis with the following organisms: Diplococcoid and short fusiform bodies, Gram positive diplococci (streptococci).

Treatment of this affection has been varied to a wide degree, but up to the present time salvarsan seems quite the favorite, whether used locally or intravenously, according to the severity of the condition.

Thus far, however, there is nothing that points the way by which we may successfully and surely bring to termination repeated attacks of so-called Vincent's angina upon the tonsil.

Salvarsan may be specific for the immediate attack, but it has little or no influence in preventing recurrences. That repeated attacks occur in the same patient is well known, and that the tonsil is probably the most frequent point of attack is generally accepted. This has been pointed out by Wherry (1911), who states that Vincent's angina is generally an affection of the tonsillar crypts and even goes so far as to locate the usual seat of the affection in the upper angle of the tonsil behind the palatal arch.

There can be no question that the usual methods of treatment are efficacious in mild attacks where there is, for instance, a small ulceration of one or both tonsils. Who is to say the patient will not have a recurrence in a more malignant form, as was reported by Deglos in the *Lyon Medical Journal* in June, 1918? Indeed, the writer has one such case, the first of the series, which, in fact, prompted this study.

These considerations have led me to undertake the removal of the tonsil both as a therapeutic and preventive measure in the active stages of Vincent's angina. I feel that although the cases are only fifteen in number, they show that tonsillectomy, in the presence of a Vincent's angina, effectually and permanently cures the disease, and that the operation is followed by no more severe reaction than that after a tonsillectomy under usual conditions. In the entire series there was no immediate or postoperative hemorrhage and no local or systemic infection discernible. Further, the bacteriologic examinations showed most interesting and marked immediate effects upon the presence of the bacterial flora. In fact, both the spirochetes and bacilli were not discoverable in the tonsil immediately after the operation, however numerous they were before.

The Vincent-Plaut bacillus, as is well known, has been found in the nose, the intestines, the liver, the ear, uterus and around the foreskin of the penis, apparently without active symptoms. It may, therefore, be assumed, as many writers have suggested, that the Vincent-Plaut bacillus in itself may create no actual pathologic process, but becomes active only when the proper media are created through the influence of other bacteria. Thus it was demonstrated in a series of 496 patients at one of the base hospitals that 196 of these showed the bacillus fusiformis and spirocheta.

In my series, smears were taken from four points in each case: the right tonsil, the left tonsil, behind the upper central incisors, behind the lower central incisors. In a small per cent of these cases we found fusiform bacilli without spirilla, but in the majority both the fusiform and spirilla were demonstrated. These were all streak preparations, no cultures being made. These patients were healthy so far as to local mouth or throat symptoms. Deglos found Vincent's organisms in 21 out of 255 patients sent to him for angina or syphilis. In a series of cases cited by Holm in 1910, in 265 cases, 99 of which were suspected diphtheria, the bacteriologic examination showed in 64 cases diphtheria was present, and out of 73 cases 35 showed fusiform bacilli. In the last two groups there was infection in the mouth, while in the first the mouths were apparently healthy. Many cases have been reported in which a secondary Vincent's angina was found superimposed upon a primary infection.

Recently the writer operated upon a patient of Dr. H. W. Loeb. This patient had been observed by other men over a period of 18 months. There was tremendous glandular involvement on the right side of the neck, with ulceration of the right tonsil, pillars and soft palate. Slides made from these areas showed numerous fusiform bacilli and spirilla. Cultures were negative; Wassermann negative. Operation was decided upon as a typical case of so-called malignant Vincent's. The laboratory showed, however, the case to be one of carcinoma.

I am in agreement with Graley, Kiefer, Lacogue and others who think the infection and consequent destruction of tissue purely a secondary affair, the primary infection providing suitable media in which the bacillus fusiformis and spirilla may thrive.

Larson and Barron (1913) recite a case in which the bacillus fusiformis and spirilla were cultured from the blood stream two days after death. While this is certainly possible, it may be purely an accidental occurrence. In toxic cases, for instance, I am convinced that the toxemia is due not to the bacillus fusiformis but to a streptococcal primary invasion or other toxic bacteria. My experience also leads me to believe that a patient may never become seriously ill from the Plaut bacillus alone

and that activity of this bacterium is purely a local one. On these premises it was consistent for me to disregard the usual surgical caution, justified in the face of most acute infections, and I was impelled to operate upon the patient at the time of the infection.

In selecting cases for tonsillectomy, the following were excluded: 1. Those in which the process was slight and the symptoms mild. 2. Those in which there was no history of recurrence, except in two cases in which the process was very extensive. 3. When the Wassermann was positive. In this way, the series is confined to the definite extensive recurrent nonsyphilitic type. In each instance the patient was kept under close observation for several days, during which the bacteria were studied from numerous smears. The tonsillectomy was in every case performed under local anesthesia, using 200 cc. of a half per cent solution of novocain, to which was added five minims of adrenalin chlorid 1 to 1,000. The tonsil was carefully freed from its bed, with a very sharp knife, and removed with a cold snare. I may add here the anesthesia seemed in all cases quite as efficient as under the noninflammatory status.

After having operated upon ten cases with good results, prompted by curiosity I took some smears from the ulceration just previous to the injection, then again from the same area immediately after the tonsil was removed from its bed. To my great astonishment, I found that while the bacilli were numerous in the first slides, the second series, taken after the tonsil was removed and from the same area, showed an entire absence of the bacillus fusiformis and spirillum. The last five cases in the series confirmed this observation, which was made by five different well known bacteriologists. Thinking perhaps the injection may have some influence upon the disappearance of the bacilli, I then made three series of smears; one from the infection before operation, one after the injection, and one after the tonsils had been removed. It was found the injection seemed to have little or no influence, as quite as many bacilli were found as before. Pilot and Braum say, in enucleation of the tonsil, granules from the crypts of the tonsils are readily lost, but this cannot account for the disappearance of the bacilli in the face of extensive ulceration.

No satisfactory solution of this strange phenomenon has thus far been made, nor has even a possible explanation been suggested by the bacteriologists who have been consulted. Further observation may discover the explanation, which might have an important bearing on the bacteriology and perhaps upon the treatment of the affection.

Appended are reports of fifteen cases operated upon with successful result as to cure and nonrecurrence.

SUMMARY*.

A study of the fifteen cases here reported suggests the following:

1. An active severe Vincent's angina is no contraindication to tonsillectomy.
2. Relief from the symptoms follows the tonsillectomy.
3. No recurrence has been observed.
4. The tonsil must be an important factor in the chronicity and recurrence of the disease.
5. The spirilla and fusiform bacilli associated with the disease almost instantly disappear after operation.
6. No satisfactory explanation of this rapid disappearance has been found.
7. This work may open up a new field for the study of the etiology of Vincent's angina.

CASE REPORTS.

Case I.—October, 1918. Male, aged 26, single, machinist.

Past history, more or less negative. Had some of the usual diseases of childhood, but on the whole has been very well. No history of venereal disease.

Present History.—Never had any throat trouble until 18 months ago, when he contracted a very sore throat on the right side, for which he was treated for about two months. He recovered from this attack, only to have another about two months later—this time on both sides. He was again treated. Recovery was much slower. He had had eight attacks before he came under my observation. At this time there had taken

*Since the completion of this paper, Dr. J. A. Morgan has reported (*Laryngoscope*, Vol. XXXIV, page 722) a single case of Vincent's angina, which was relieved by tonsillectomy.

place great destruction of the tonsillar tissue, both anterior and posterior pillars, including the soft palate on both sides, and he was suffering from a bilateral ulceration of the tonsils. He was treated for ten days with excellent results, remaining well for a week, when he returned with condition as bad as before.

Wassermann negative.

Operation was advised and performed, with little or no reaction except the ordinary postoperative one.

Previous to the operation, the slides made from the ulcerations were rich with fusiform bacilli and spirilla, while after the operation slides from the fossa showed:

First day after operation—		
Fusiform bacilli	*	
Spirilla	*	
Second day after operation—		
Fusiform bacilli	*	
Spirilla	†	
Third day after operation—		
Fusiform bacilli	†	
Spirilla	†	
Fourth day after operation—		
Fusiform bacilli	*	
Spirilla	†	
Cocci	*	
Fifth day after operation—		
Fusiform bacilli	†	
Spirilla	†	
Cocci	*	

The patient made an uneventful recovery and had no subsequent attacks for 13 months, after which he passed from my observation. Examinations for fusiform bacilli and spirilla were all made by streak method. No attempt was made to culture spirilla.

Case II.—December, 1918. Male, aged 24, single, clerk.

Past History.—Had always been in perfect health until four months previous. There is nothing of special importance in the past history.

Present History.—Developed a sore throat suddenly four months previous, which became rapidly worse. Both tonsils became ulcerated and, according to his story, his physician cauterized them with medicine. After several treatments, patient improved, though his throat never felt quite right. Three weeks later, the right side of throat became infected with the same type of ulcer, and three or four days later the left side became involved in spite of treatment. He recovered from this, the second attack, in about two weeks of constant treatment.

The patient came to me in November, 1917. Both tonsils were badly ulcerated. There was also some stomatitis about the lower teeth.

Wassermann negative.

Smears from each tonsil and mucosa of the mouth showed fusiform bacilli and spirilla in great numbers.

Diagnosis of Vincent's angina was made and the tonsils removed the fourth day. No local treatment was given in the meantime. The patient made an uneventful recovery, with no particular reaction other than the usual one of postoperative tonsillectomy.

FINDINGS.

Slide preparations first day after operation—

Fusiform bacilli	*
Spirilla	*
Bacteria	†
Cocci	*

Second day after operation—

Fusiform bacilli	*
Spirilla	†
Bacteria	‡
Cocci	‡

Third day after operation—

Fusiform bacilli	*
Spirilla	†
Bacteria	‡
Cocci	*

Fourth day after operation—

Fusiform bacilli	†
Spirilla	†
Bacteria	*
Cocci	†

Fifth day after operation—

Fusiform bacilli	†
Spirilla	†
Bacteria	*
Cocci	*

Operation was performed as usual, and the patient was discharged and resumed duty in a few days. He was observed from time to time over a period of 13 months, with no subsequent trouble with the throat or mouth.

Case III.—February, 1919. Male, aged 26, unmarried, farmer.

Past History.—Pneumonia twice, typhoid fever, diphtheria, mumps. Has been perfectly well since boyhood, with the exception of several attacks of tonsillitis in the past few years. Looks robust and in excellent health now.

Present History.—A year and a half ago he suffered from a severe attack of sore throat, which caused him to quit work, though he did not feel bad generally. His throat, however, was very sore, and the tonsils were ulcerated. He was treated by local physician with mouth washes, gargles, etc. After two weeks of such treatment the doctor burned the ulcers twice, after which marked improvement and recovery followed. Two months later, however, he had a severe recurrence. Another physician then told him he had syphilis and gave him an injection, and his throat improved in a few days.

I saw him in the fourth attack. At the time both tonsils were much involved, large and deep ulceration in each, with but little reaction in the surrounding tissue. The patient did not feel ill nor did he complain particularly of discomfort. Temperature normal.

Wassermann negative.

He was given a mild mouth wash, after which slides were taken, which were found to be rich in fusiform bacilli and spirilla. Slides taken for four days showed practically the same picture.

Diagnosis: Vincent's angina. Operation on the fifth day. Very little reaction and uneventful recovery.

FINDINGS.

Slide preparations made:

First day after operation—

Fusiform bacilli	*
Spirilla	†
Bacteria	*

Second day after operation—

Fusiform bacilli	†
Spirilla	†
Bacteria	‡

Slides taken on the third, fourth and fifth days after operation failed to reveal either fusiform bacilli or spirilla.

Patient was discharged to resume duty. There has been no recurrence over a period of two and one-half years.

Case IV.—March, 1919. Male, aged 28, single, mechanic.

Past History.—An orphan. Had never been ill, so far as he knows, until two years ago, when he had a very sore throat for three weeks with great patches on the tonsils. He had no treatment except salt and vinegar gargle. He recovered apparently after two weeks. Six months later, he had another attack similar to the first one. A physician at this time told him he had ulcerative tonsillitis and burned out the tonsils and the patient recovered in about ten days.

Present History.—Since entering the Army he had two more attacks of the same disease. I saw him in the fifth attack. Both tonsils were badly ulcerated, some scar tissue in the surrounding structure. Patient looks ill, temperature normal, no adenitis.

Wassermann negative.

Slides from each tonsil show great numbers of bacilli fusiformes and spirilla. Operation on the fourth day. Patient stood the operation fairly well but was very nervous. He went to bed feeling badly. Recovery, however, was rapid and with no complications except the usual postoperative discomfort.

FINDINGS.

Slide preparations from each fossa:

First day after operation—	
Fusiform bacilli	*
Spirilla	*
Bacteria	*
Cocci	*
Second day after operation—	
Fusiform bacilli	*
Spirilla	†
Bacteria	*
Cocci	*
Third day after operation—	
Fusiform bacilli	†
Spirilla	†
Bacteria	*
Cocci	*
Fourth day after operation—	
Fusiform bacilli	†
Spirilla	†
Bacteria	*
Cocci	†
Fifth day after operation—	
Fusiform bacilli	†
Spirilla	†
Bacteria	*
Cocci	†

Patient discharged for duty. One month later he was observed again and had been perfectly well. After that time, however, he was lost from observation.

Case V.—March, 1919. Male, aged 22, unmarried, clerk.
Past History.—Negative.

Present History.—Had been in excellent health until four months ago, when he developed a very sore throat on the right side. I saw him a few days after the onset, and found a deep ulcer about 4 mm. in diameter on the right tonsil, with little or no reaction in the surrounding tissue.

Slides taken were found to be rich in fusiform bacilli and spirilla.

Wassermann negative.

He was treated for seven days with chromic acid daily applied direct to the ulceration, and recovered nicely. Two weeks later he came in with another attack on the same side, as bad as before. Again smears were taken and a diagnosis of Vincent's angina was made.

Patient in the meanwhile continued his work. Ninety per cent silver nitrate was used, this time daily for ten days. Patient steadily improved, and was discharged on the tenth day. Six weeks later he was observed again in another attack. This time both tonsils were involved. Observation was made for three days, without treatment, ulcerations growing steadily worse. Operation was performed on the fourth day. The patient stood the operation very well, having no pain during the procedure, and made an uneventful recovery.

FINDINGS.

Slide preparations from each fossa:

First day after operation—

Fusiform bacilli	*
Spirilla	†
Bacteria	*
Cocci	‡

Second day after operation—

Fusiform bacilli	*
Spirilla	†
Bacteria	‡
Cocci	‡

Third day after operation—

Fusiform bacilli	‡
Spirilla	†
Bacteria	*
Cocci	*

Fourth day after operation—

Fusiform bacilli	‡
Spirilla	†
Bacteria	*
Cocci	*

Fifth day after operation—

Fusiform bacilli	†
Spirilla	†
Bacteria	*
Cocci	*

Sixth day after operation—

Fusiform bacilli	†
Spirilla	†
Bacteria	*
Cocci	†

Patient was discharged as recovered. He had no subsequent attacks in three years.

Case No. VI.—August, 1919. Male, aged 20, single, railroad clerk.

Past History.—From childhood he remembers having had attacks of sore throat, frequent colds, and earache with discharge from ears. Had several childhood diseases, but does not remember what they were.

Present History.—Seven days ago developed a sore throat on the right side, which was getting worse. When I saw him he had a deep ulcer of the right tonsil, but no involvement of the surrounding structure. Patient felt fairly well except on swallowing. Ulcer in the center of the tonsil and about 5 mm. in diameter.

Slides taken were found to be rich in fusiform bacilli and spirilla. He was treated with 75 per cent silver nitrate applied direct to the ulceration, and after the third day the ulceration was healed.

Two weeks later he appeared again with an ulcer in both tonsils. Both were centrally located, the right one being more extensive.

Smears showed numerous fusiform bacilli and spirilla from both ulcers.

Wassermann negative.

Diagnosis: Vincent's angina.

No treatment was given him, and the ulceration progressed until the fifth day, when both tonsils were removed. The pa-

tient made an uneventful recovery, with little reaction other than that which usually follows tonsillectomy.

FINDINGS.

Slide preparations from each fossa:

First day after operation—	
Fusiform bacilli	*
Spirilla	*
Bacteria	*
Cocci	†
Second day after operation—	
Fusiform bacilli	*
Spirilla	*
Bacteria	†
Cocci	*
Third day after operation—	
Fusiform bacilli	*
Spirilla	†
Bacteria	†
Cocci	†
Fourth day after operation—	
Fusiform bacilli	*
Spirilla	†
Bacteria	*
Cocci	†

This patient has had no recurrence in four years.

Case VII.—July, 1920. Male, aged 28, married, railway conductor.

Past History.—Of no special importance. Has been in very good health all his life.

Present History.—For more than a year has had repeated attacks of sore throat, which persisted from ten days to three weeks. The present attack began four days ago. Patient is very uncomfortable; swallowing painful; getting rapidly worse. Previous attacks have been of similar character, but usually on one side only. On examination, he presents a deep ulcer on each tonsil, the right one being in the center, the left one in the upper portion. There is little or no involvement of the surrounding structure and patient is able to perform his duties.

Diagnosis: Vincent's angina (both tonsils rich in fusiform bacilli).

Wassermann negative.

Operation the third day after his first visit. Patient reacted normally to operation. No pain; no complications.

FINDINGS.

Slide preparations:

First day after operation—

Fusiform bacilli	*
Spirilla	†
Bacteria	*
Cocci	†

Second day after operation—

Fusiform bacilli	*
Spirilla	†
Bacteria	†
Cocci	*

Third day after operation—

Fusiform bacilli	†
Spirilla	†
Bacteria	*
Cocci	*

Fourth day after operation—

Fusiform bacilli	†
Spirilla	*
Bacteria	†
Cocci	*

Fifth day after operation—

Fusiform bacilli	†
Spirilla	†
Bacteria	*
Cocci	*

Sixth day after operation—

Fusiform bacilli	†
Spirilla	†
Bacteria	†
Cocci	*

Smears made from each tonsil just previous to operation showed numerous fusiform bacilli and spirilla. In the smears made again from the ulceration within three minutes after the tonsils had been removed, nothing could be found over this area except pus cells, a few micrococci and bacteria, which on culture proved to be staphylococci.

The patient has had no subsequent attacks over a period of three and one-half years.

Case VIII.—June, 1920. Male, aged 27, married, railroad switchman.

Past History.—Has had many attacks of tonsillitis since childhood. So far as he knows, he never had an abscess. Does not remember much of his early life or parents, but has generally been healthy.

Present History.—About a week ago he developed a sore throat on the left side which grew rapidly worse. Three days later he noticed that both sides were sore. Examination shows deep and extensive ulceration of both tonsils extending to the anterior pillar of the left side, with considerable reaction of the surrounding tissue, but with a distinct line of demarcation. Smears taken from each tonsil show large numbers of spirilla and fusiform bacilli.

Wassermann negative.

For two successive days slides were made, which showed large numbers of fusiform bacilli and spirilla. On the fourth day, operation was performed. Patient had not been ill at any time and was able to continue his work up until the day of the operation. Temperature normal. The patient withstood the operation very well, with nothing but the usual postoperative reaction.

FINDINGS.

Smear preparations from each fossa:

First day after operation—

Fusiform bacilli	†
Spirilla	†
Bacteria	†
Cocci	*

Second day after operation—	
Fusiform bacilli	*
Spirilla	†
Bacteria	*
Cocci	*
Third day after operation—	
Fusiform bacilli	*
Spirilla	†
Bacteria	*
Cocci	*
Fourth day after operation—	
Fusiform bacilli	†
Spirilla	†
Bacteria	*
Cocci	*
Fifth day after operation—	
Fusiform bacilli	†
Spirilla	†
Bacteria	*
Cocci	†

Patient was discharged. There has been no recurrence over a period of two and one-half years' observation.

In this case smears were made directly previous to the operation and found to be rich in fusiform bacilli and spirilla. Smears from each ulcer of the tonsils, within five minutes after removal, showed few fusiform bacilli and no spirilla.

Case IX.—May, 1921. Male, aged 25, clerk.

Past History.—More or less negative. Has never been particularly robust at any time; always underweight and has had several diseases of childhood, none of which were serious.

Present History.—For the past ten days he has been suffering from a severe sore throat, with great difficulty in swallowing. Condition first became bad on the right side, and two days later the left side became involved. Has had several attacks of sore throat, but none as bad as the present one, and probably none of the same type. He appears now with extensive deep ulcers on both tonsils, with considerable reaction in

the surrounding structure. Pain radiates to both ears and more or less continuous. Patient feels ill and has not been able to work; does not sleep well at night; appetite is gone. Cervical glands on both sides are swollen, and also the sublingual glands. Temperature is normal.

Diagnosis: Vincent's angina, which is verified by smears from each tonsil, which are especially rich in fusiform bacilli and spirilla. Cultures show staphylococci and pneumococci.

Wassermann negative.

Four days' observation, during which time destruction of the tonsils rapidly progressed. Operation on the fourth day. Reaction from the operation more than usual in this case, and the patient was kept in bed for two days, after which time he steadily improved. At no time, however, did he have a rise of temperature. White cell count was 98,000 the second day after operation. He resumed his usual occupation on the sixth day after operation and felt particularly well, except for some soreness of the throat on the left side due to granulation along the margin of the wound.

FINDINGS.

Slide preparations:

First day after operation—

Fusiform bacilli	‡
Spirilla	*
Bacteria	*
Cocci	‡

Second day after operation—

Fusiform bacilli	‡
Spirilla	*
Bacteria	‡
Cocci	‡

Third day after operation—

Fusiform bacilli	*
Spirilla	†
Bacteria	‡
Cocci	*

Fourth day after operation—

Fusiform bacilli	*
------------------------	---

Spirilla	†
Bacteria	†
Cocci	*
Fifth day after operation—	
Fusiform bacilli	†
Spirilla	†
Bacteria	*
Cocci	†
Sixth day after operation—	
Fusiform bacilli	†
Spirilla	†
Bacteria	*
Cocci	†

No recurrence over a period of one and one-half years.

Case X.—Female, aged 21, single, clerk.

Past History.—Has always been healthy except for the usual diseases of childhood. No serious illness.

Present History.—Seven days ago throat became sore, gradually getting worse. Considerable pain on swallowing. Never had such condition before. Not subject to tonsillitis. Examination of the left tonsil showed a deep ulcer covering about one-half of the structure. There is little involvement of the surrounding tissue. Patient does not feel ill.

Smears show Vincent's angina.

Wassermann not made.

Tonsils removed the third day after admission to the hospital. Patient stood operation well, there being no complications except a slight bleeding on the second day from the right side. Blood clot was removed from this fossa and bleeding controlled by pressure.

This patient ran a temperature of 102 on the second day, which went to normal, however, the following morning. She was discharged on the fourth day.

The bacteriologic chart in this case shows smears made three times daily from the second and third day following operation. On the fourth day smears were made at 8 a. m. and 8 p. m., and on the fifth and sixth days smears were made only once.

	R. Tonsil	L. Tonsil	Palate	Teeth
Aug. 3, 1921 7:30 p. m.	Bacteria ‡ Str. *	Fusiform ‡ Spirilla ‡	Diplo- cocci †	Spirilla • Fusiform ‡ Bacteria ‡ Cocci ‡
10 p. m.	Bacteria ‡ Str. • Cocci ‡	Spirilla ‡ Fusiform ‡ Bacteria ‡ Cocci ‡	Cocci ‡ Spirilla ‡ Fusiform ‡	Bacteria ‡ Cocci ‡ Spirilla ‡ Fusiform •
Aug. 4, 1921 8 a. m.	Bacteria † Cocci †	Spirilla • Fusiform ‡	Strep. † Staph. † Bacteria †	Spirilla • Fusiform •
9 a. m.	Cocci †	Fusiform ‡ Spirilla ‡	Cocci †	Fusiform ‡ Spirilla ‡ Cocci ‡ Bacteria •
9:10 a. m.	Cocci •	Cocci •	Cocci •	Cocci • Bacteria •
Operation After opera- tion	Little or nothing	Little or nothing		
Aug. 4, 1921 12 Noon	Few bacteria			Bacteria ‡ Cocci ‡
3 p. m.	Cocci •	Cocci •		Bacteria ‡
6 p. m.	Cocci • Bacteria •	Bacteria ‡		Cocci •
Aug. 5, 1921 8 a. m.	Bacteria ‡	Spirilla ‡ Fusiform ‡ Bacteria ‡		Bacteria ‡
11 a. m.	Spirilla † Fusiform † Bacteria •	Spirilla † Fusiform † Bacteria ‡		Bacteria †
4 p. m.	Spirilla † Fusiform † Bacteria †	Spirilla • Fusiform • Bacteria †		Bacteria ‡
Aug. 6, 1921 8 a. m.	Spirilla • Fusiform † Bacteria ‡	Spirilla • Fusiform • Bacteria •		Bacteria †
8 p. m.	Spirilla • Fusiform • Bacteria ‡	Spirilla • Fusiform • Bacteria ‡		Bacteria ‡
Aug. 8, 1921	Spirilla † Fusiform † Bacteria †	Spirilla † Fusiform † Bacteria •		Bacteria ‡
Aug. 9, 1921	Spirilla † Fusiform † Bacteria •	Spirilla † Fusiform † Bacteria †		Bacteria ‡

Case XI.—June, 1923. Male, age 21.

Past History.—Has always been in good health except for the usual diseases of childhood, none of which was serious. Never had any throat trouble until a month ago.

Present History.—About a month ago developed a very sore throat on the left side, which was treated for four or five days with good results. One week later his throat again became sore on the corresponding side, at that time presenting a deep ulcer of grayish appearance, in the left tonsil, with no inflammatory reaction of the surrounding structure.

Smears were positive for Vincent's angina.

Patient was treated with 50 per cent silver solution, and after three days was discharged as cured. He remained well for nearly a month, when he returned with a bilateral ulceration. Both ulcers were very deep and quite extensive. Smears again showed Vincent's angina.

Wassermann previously done—Negative.

Operation was performed the third day after the onset of the third attack. Patient made an uneventful recovery, with very little discomfort, and returned to work on the fifth day after operation.

FINDINGS.

Slide preparations:

First day after operation—

Fusiform bacilli	†
Spirilla	†
Bacteria	*

Second day after operation—

Fusiform bacilli	†
Spirilla	*
Bacteria	†
Cocci	*

Third day after operation—

Fusiform bacilli	*
Spirilla	†
Bacteria	†
Cocci	*

Fourth day after operation—

Fusiform bacilli	†
Spirilla	†
Bacteria	*
Cocci	*

Fifth day after operation—

Fusiform bacilli	†
Spirilla	†
Bacteria	*
Cocci	†

Smears were made from each tonsil just previous to operation and were found to be rich in fusiform bacilli and spirilla.

Smears made from each tonsil after removal show no fusiform bacilli, no spirilla, very few cocci and few bacteria.

This patient has had no subsequent attacks over a period of fifteen months.

Case XII.—June, 1923. Male, aged 20, clerk.

Past History.—Negative. No bearing on present illness.

Present History.—Patient presents himself with a small ulcer on the right tonsil. Has had a sore throat for three days. Ulcer is about 3 mm. deep and confined to the upper pole. There is considerable inflammatory reaction about the surrounding structure, and patient appears ill.

Smears made show many fusiform bacilli and spirilla.

Patient grew progressively worse, in spite of treatment, and on the fourth day the ulcer involved a much greater portion of the right tonsil. Anterior cervical glands swollen, patient feeling quite ill. Temperature 100. Two days later superficial necrosis entire face of the right tonsil, left side now becoming involved.

Smears still show Vincent's organisms and spirilla on the right side.

Wassermann negative.

Diagnosis: Malignant or progressive Vincent's angina.

Operation on the seventh day. From a nervous standpoint, the patient reacted badly to operation. In no other way was there anything unusual. He made a complete and rapid recovery. On the fourth day the glands had subsided. The first day after operation the patient's temperature was $100\frac{1}{2}$ and remained so the entire day, the white cell count being 11,000.

On the second day temperature was normal and patient left hospital. On the sixth day he resumed his work.

FINDINGS.

Slide preparations:

First day after operation—

Fusiform bacilli	†
Spirilla	*
Bacteria	†
Cocci	†

Second day after operation—	
Fusiform bacilli	†
Spirilla	†
Bacteria	†
Cocci	†
Third day after operation—	
Fusiform bacilli	*
Spirilla	†
Bacteria	†
Cocci	†
Fourth day after operation—	
Fusiform bacilli	*
Spirilla	†
Bacteria	†
Cocci	*
Fifth day after operation—	
Fusiform bacilli	*
Spirilla	*
Bacteria	†
Cocci	†
Sixth day after operation—	
Fusiform bacilli	†
Spirilla	†
Bacteria	†
Cocci	†

This was the most serious case in the series. There has been no recurrence in a period of fourteen months.

Smears taken from the ulcerated areas just previous to operation were rich in fusiform bacilli and spirilla. Slides taken from the tonsil just after removal show occasional fusiform bacilli, no spirilla, few bacteria and few cocci.

Case XIII.—December, 1923. Male, aged 37, single, clerk.

Present History.—Has had trouble with the right side of throat for seven months. Has been quite sore at times, with intervals of relief, though, on the whole, condition is getting worse, in spite of treatment by several physicians.

Examination shows extensive ulceration of right tonsil, anterior and posterior pillars, and buccal membrane posterior. The cervical, sublingual and maxillary glands are involved to a great extent. Patient has not lost weight; looks quite ro-

bust. There is nothing on the left side. Smears made from the affected area show great numbers of fusiform bacilli and spirilla.

Wassermann negative.

Patient was observed and treated locally for seven days, when it was decided to remove the tonsils. Reaction to tonsillectomy was slight in every way. The fossæ healed rapidly. The surrounding structures, however, did not improve nor did the operation have any effect on the glands. Sections made from the right tonsil proved it to be carcinoma, which perhaps originated in the tonsil.

Note.—One year later patient was reported as in a dying condition.

Diagnosis previous to operation: Progressive Vincent's angina.

Diagnosis subsequent to operation: Carcinoma of the right tonsil.

Case XIV.—April, 1924. Male, aged 25, married, machinist.

Past History.—Of no importance; has been generally well.

Present History.—About two weeks ago developed very sore throat on left side, which has grown rapidly worse until now, and in spite of treatment, there is a deep, sloughing ulcer in the right tonsil involving nearly all of that structure. There is very little inflammatory reaction of the adjacent structures, and patient feels quite well except for sore throat and pain radiating to the left ear. Right side not involved. Tonsillectomy was performed on the sixth day under local anesthesia, patient reacting well, with no complications. This patient ran a temperature of 100 on the day of operation, though temperature was normal when admitted to the hospital. On the second day temperature was 99, the third day 99½, and the fourth day normal. He was discharged on that day.

FINDINGS.

Slide preparations:

First day after operation—

Fusiform bacilli	*
Spirilla	†
Bacteria	*
Cocci	*

Second day after operation—	
Fusiform bacilli	*
Spirilla	†
Bacteria	*
Cocci	*
Third day after operation—	
Fusiform bacilli	†
Spirilla	†
Bacteria	*
Cocci	*
Fourth day after operation—	
Fusiform bacilli	†
Spirilla	†
Bacteria	*
Cocci	*
Fifth day after operation—	
Fusiform bacilli	†
Spirilla	†
Bacteria	*
Cocci	*

Smears taken from the ulceration of the tonsil just previous to operation were rich in fusiform bacilli and spirilla. Smears taken from the ulceration of the tonsil just after injection showed fusiform bacilli numerous and spirilla numerous. Smears taken from the ulceration of the tonsil after the tonsil had been removed showed an entire absence of fusiform bacilli and spirilla.

Case XV.—April, 1924. Male, aged 30, married, railway conductor.

Past History.—Unimportant. Patient has been healthy all his life.

Present History.—For the past four days has had severe sore throat on both sides. Was examined by my associate, who made a diagnosis of acute follicular tonsillitis, prescribed sodium bicarbonate gargle, together with empirin compound, 5 grains every two hours. Patient appeared two days later with deep and extensive ulceration of right tonsil, involving the entire surface of that structure. He feels fairly well, except

for pain on swallowing, and there is very little reaction of the surrounding tissue and no palpable glands.

Smears made from each tonsil were rich in fusiform bacilli and spirilla. Smears were taken each day for four days. All showed great numbers of fusiform bacilli and spirilla.

Wassermann negative.

Patient was operated on the fourth day.

Ulcerations extended to both anterior pillars. Temperature normal. Patient stood operation very well. There was no unusual reaction, and healing was rapid and uneventful. Ulcerations on pillars disappeared the third day.

FINDINGS.

Slide preparations:

First day after operation—

Fusiform bacilli	*
Spirilla	*
Bacteria	*

Second day after operation—

Fusiform bacilli	†
Spirilla	†
Bacteria	†
Cocci	*

Third day after operation—

Fusiform bacilli	†
Spirilla	†
Bacteria	†
Cocci	*

Fourth day after operation—

Fusiform bacilli	*
Spirilla	*
Bacteria	†
Cocci	†

Fifth day after operation—

Fusiform bacilli	†
Spirilla	†
Bacteria	*

Smears made from the tonsil just before operation showed many fusiform bacilli and spirilla present. Smears made from

the tonsil just after injection showed many fusiform bacilli and few spirilla. Smears made again from the ulcerated portion within five minutes after tonsils were removed showed no fusiform bacilli or spirilla in the tonsil.

This patient has had no subsequent attacks during a period of seven months.

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XCIII.

MALIGNANCY OF THE EXTERNAL NOSE WITH REPORT OF CASE OF X-RAY ORIGIN.*

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A review of the literature on malignancies of the external nose is not rewarded by a very voluminous amount of material. Perhaps the dearth of reports may be accounted for by the general assumption that the external nose shows the same type of neoplastic change characteristic of epithelioma elsewhere in the body. Although this is true in a general way, the epithelium of the nose is singular in that it is stretched rather snugly over a projecting structure and covers little or no subcutaneous soft tissue. The skin of the nose is constantly exposed to natural trauma and atmospheric agents, not to mention extraneous elements, such as intensive artificial lights, alpine lamps, and ultraviolet and X-rays.

Practically every type of malignant cutaneous lesion is now generally conceded to have arisen as a result of chronic irritation. Various gases and chemicals have been named as causative factors, but as these agents are encountered only in certain industries and as cancer is almost universal, the theory of their etiologic importance has not gained much credence. Of all the mechanic or biochemic processes conducive to the development of malignancy, probably the most insidious are those introduced by man in his efforts at therapeusis of relatively harmless and benign conditions.

It is a distressing fact that malignant neoplasms sometimes result from overdosage during the treatment of skin or other lesions by the X-ray. Such disastrous effects are usually consequent to the administration of X-ray treatment by a technician who is not under the direct supervision of a responsible radiologist.

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The indiscriminate use of the X-ray by untrained operators in the treatment of tonsillar disease, eczemas, deafness, and a number of none too well understood conditions is fraught with more danger than is generally imagined and should be vehemently condemned. The epidermis, particularly of the hands and face, is very susceptible to the X-rays, and an epithelioma resulting from overexposure, either from small doses over a long period of time or larger doses over a shorter period, will not yield to X-ray therapy.

The possibility of collecting cases of carcinoma developing on benign lesions treated with the X-ray is rather remote, in view of the unfavorable light that would be thrown on the author-confessor of any such article. Vogt, however, has succeeded in collecting cases of carcinoma developing in gynecologic conditions, which were probably started by X-ray treatment. From a survey of the literature and cases observed by practitioners, he found:

One case of vulvar carcinoma after superficial irradiation, one of the abdominal walls after deep therapy, six cases of ovarian carcinoma after deep therapy, nineteen cases of uterine cancer (of which sixteen affected the corpus, three the cervix) and a case of double sided mammary cancer. Of these Vogt dismisses all but three, viz., those of the vulva, the abdominal wall and the mammae.

Another agent which has come into much prominence in recent years and carries possibilities of mischief not before considered is the ultraviolet ray. We see on every side elaborate equipment for treatment of all and every sort of malady by ultraviolet light, and in most cases this therapy is in the hands of lay assistants, who carry on their work by rule of thumb rather than with any understanding of the fundamental structures and effects produced. It is not unlikely that in the future we may see increasing numbers of cutaneous lesions traceable to overdosage.

Finsen, in his early work, described the granular changes in the rete malphigian cells due to the irritant effect of such lamps. Cheatele called attention to the fact that some skins show atrophic changes due to exposure to sunlight. He named the condition "biotripsis," and remarked that this change in the skin furnishes a common site for squamous epithelioma. Gar-

deners, as might be expected, are frequent subjects for squamous epithelioma on the back of the hand. The ultraviolet ray has also been found to hasten the advent of carcinoma on the skin in mice which have been painted with tar, and also that it encourages growth once the tumor has developed. Cheate himself observed a carcinomatous lesion become steadily worse on exposure to ultraviolet rays. Bunting has recently observed a child severely burned by ultraviolet rays, and is of the opinion that they are potentially dangerous.

The potential harmfulness of the X-rays and ultraviolet rays in no way condemns them as therapeutic agents, but rather emphasizes the need for judicious application. The following case is reported as a caution against permitting ignorant or partially educated persons to use these powerful agents, which require a well seasoned knowledge for the proper direction of their bifold powers.

REPORT OF CASE.

Case 37230. Miss A. R., Norwegian, aged 29 years, came to the Jackson Clinic September 7, 1923. Her mother, who died while the patient was a small child, was said to possess a very sensitive skin. There is one brother and one sister, who apparently possess normal skins.

The patient, as a child, was reported to have had a very white skin, which apparently was easily irritated by exposure to the sun. When she was six years of age a rash appeared on the arm and spread until it covered the entire body. This has persisted more or less to date, especially on the face. When it first appeared it was diagnosed as eczema. Even at the time of her admittance to the hospital the patient would develop a pronounced erythema on exposure to sunlight for half an hour.

At the age of 16, or in 1909, her relatives were advised to have her treated with X-ray, as they had heard that an X-ray machine was established in Christiania and that treatments were being given for all sorts of diseases with wonderful results. She was taken to Christiania, and over a period of thirty days was given daily treatments, the face being exposed to the rays five to ten minutes on each side. There was apparently some decrease in extent of the rash for seven weeks, but it reappeared, and the following year a heavier dosage was ad-

ministered. From the age of 18 to the time of her admission to the Jackson Clinic she had frequent and repeated treatments with X-ray.

The patient came to the United States in 1914, and a short time later went to a physician in Milwaukee, who diagnosed her condition as one lacking in skin pigment layer and gave her the first bit of sane advice she had received. He told her to "take no more treatments." She failed to follow this counsel, however, and as she went about from one physician to another she continued to receive X-ray treatments varying in intensity and duration.

Operation for appendicitis was performed in 1923, after which the skin seemed to improve. In the meantime, however, what was diagnosed as a boil appeared on the tip of the nose; this was opened and some purulent (?) discharge was expressed. The lesion never completely healed, and the patient stated that the site of the boil was always uncomfortable. A red spot marked the tip of the nose. In the spring of 1923 an ulcer appeared on the site of the boil; she immediately took X-ray treatment, and the lesion increased in size. It was then treated with silver nitrate and Peruvian balsam, and while it did not become markedly worse, it certainly did not improve.

In August, 1923, a skin graft was applied, but with no beneficial result, and by October the growth was half the size of a dime. A specimen was sent for microscopic examination, and diagnosis of prickle cell carcinoma was returned. Three small doses of radium were given and the lesion healed for six weeks. It again returned and progressed like wildfire, so that in April the whole tip of the nose was involved and amputation was advised. This was not acceptable to the patient, but in June she returned and requested the operation. She was told that it was a very hopeless procedure, but she felt that something should be done either to cure or hurry the process along. In the meantime more radium was applied, but only resulted in a flare-up.

In June the nose was amputated; the incision was carried well beyond the line of demarcation and included most of the upper lip. Part of the cartilaginous septum was also removed. Immediate convalescence was good, but it was soon apparent that the growth had already extended, and small nodules ap-

peared in the lip and along the margin, especially on the left. Under chloroform anesthesia, I then instituted electrical desiccation, with low amperage and high voltage. On repeated occasions the needle was inserted deep into the tissue and current turned on until the flesh was cooked white. About the only thing accomplished was to render the growth less painful. It was perfectly evident that nothing could stay the progress of the cancer. Small glands began to appear in the preauricular and submental regions, and the mucous membrane lining of the nasal chamber was also invaded.

About this time a photograph of the patient was taken under a high power lamp, and even the few seconds' exposure necessary for this was followed by violent reaction of the whole area. The progress from this time forward was one of gradual extension. The patient was admitted to the University Hospital at Madison, and the treatment consisted of opiates to relieve pain until the end.

The growth extended up between the eyes and involved the floor of the orbit on both sides and crowded the eyes laterally. The upper lip literally dropped out, and one could look into both antrums, ethmoids and pharynx. Mastication was almost impossible, and the patient became very much emaciated. She finally died December 24, 1924. Autopsy revealed extensive involvement of the tissue adjacent to the lesion, but no metastasis could be demonstrated. The lungs showed extensive pneumonia, which was the cause of death.

PATHOLOGY.

Malignant neoplasms of the skin of the nose may be divided into two main groups: (1) Basal cell carcinoma, and (2) prickle cell carcinoma or epithelioma. The types of nasal carcinoma most readily recognized and most frequently reported are the first named. Such a growth is characterized by the usual pearly border and slow development, without metastasis. Nicholson states that a basal cell carcinoma of the epidermis is, after all, nothing more than an epithelial neoplasm, the majority of whose cells proliferate indefinitely, after the manner and in the form of the germinal cells of the rete malpighii, with very slight differentiation. This is commonly called rodent ulcer. The second type of neoplasm, or carcinoma spino-

cellulare, is much less commonly observed and of an entirely different character. It is commonly called prickle cell carcinoma or epithelioma.

Basal cell carcinoma may appear upon any skin surface, but occurs mostly commonly on the nose and forehead. In a study of 2,000 specimens of carcinoma of the skin, Broders found 13.4 per cent to be so-called rodent ulcers, occurring most frequently on the nose, eyelids, forehead and cheek. The lesion is usually solitary, but in one case, a man whose blood picture showed lymphatic leucemia, I have observed two distinct tumors, one on the nose and the other on the cheek.

The rodent ulcer appears first as an elevated whitish nodule or patch covered with scales which tend to drop off. It is essentially a slow process, but sooner or later an ulcer forms, with very little tendency to heal at the base. It is usually superficial, but in time it may become very extensive and even erode bone and cartilage. A crust may form over the dirty, necrotic base, and ulceration develops when the cells become so far removed from their source of nutriment that necrosis takes place. Additional nutrition is then supplied to the surviving cells and the lesion may extend rapidly. The extension of the lesion may produce distressing deformities of the whole region, as when the nose, cheek and upper lip are destroyed.

Green states that rodent ulcers are not important, from the point of view of malignancy, but I have found that one section of the excised ulcer is not sufficient to rule out malignancy. If the ulcer is examined in serial sections, it is surprising to find that many of these so-called unimportant or benign ulcerations will show somewhere in their border definite carcinomatous tendencies, if not true malignant changes.

According to McFarland, if the multiplications of the stratified epithelium are largely restricted to the cells of the basal layer, the tumor supposedly has its origin in them. If the basal cells multiply without transformation, the tumor is characterized by masses of similar appearing cells, and we have the carcinoma basocellulare. If, on the other hand, the growth is characterized by a disposition of the multiplying cells to complete the natural differentiation and pass through the prickle cell stage to that of true keratinization, we have the carcinoma acanthous or spinocellulare. When the prickle cells

are most abundant we have simple carcinoma spinocellulare, and when the keratin formation is most evident we have what McFarland terms "keratoides." In reality the apparently different forms are simply varying degrees of the same process. Keratinization may be seen in the form of pearls. The pearly bodies are almost as diagnostic of carcinoma spinocellulare as the prickle cells themselves.

McFarland gives a most comprehensive description of the microscopic appearance and findings. Extending, ramifying processes of epithelial elements are interwoven like the roots of a plant penetrating in all directions. In some places the epithelial processes are blunt and rounded, with the cells regularly arranged; in other places they appear as long slender columns, in which the cells are more or less disconnected, with terminal detached single cells that infiltrate the surrounding tissue. The cells of the outer layer correspond to basal cells, those of the internal layer to prickle cells, and those deep in the interior correspond to the most superficial cells of the epidermis.

TREATMENT.

The treatment of carcinoma spinocellulare consists in wide excision, either by knife or cutting cautery. If the lesion is small, the edges of the wound may be approximated with quite satisfactory results. If the denuded area is large, skin grafting may be resorted to later. Radium has failed to give encouraging results in my hands. In cases of X-ray origin the whole face may be on the verge of carcinomatous change, and any manipulation or treatment is futile, as new growth will spring up at the margin.

SUMMARY.

The epithelial structures of the skin, particularly the face and hands, are especially susceptible to the X-rays. A case is reported in which repeated exposure to the X-rays precipitated malignant changes in the skin over the nose, with a resulting extensive carcinoma of the prickle cell type. The ultraviolet light may also be held responsible for initiating, or at least encouraging, malignant changes in a benign condition.

All rodent ulcers of the nose should be regarded as potentially malignant. Microscopic examination of a single specimen is not sufficient to rule out neoplasm, as examination of serial sections throughout the mass usually reveals areas of malignant change manifested by characteristic pearl formation.

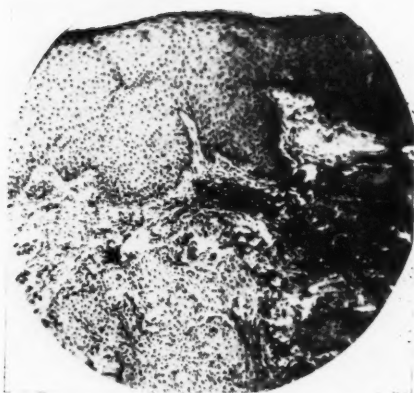
Growths characterized by a disposition of the multiplying cells to complete the natural differentiation and pass through the prickle cell stage to that of keratinization are true carcinoma spinocellulare. For all practical purposes the various microscopic pictures in which either prickle cell formation or keratinization may predominate may be regarded simply as degrees of one highly malignant process.

Treatment consists in early and wide excision of the lesion by knife or cautery. Lesions of X-ray origin are highly malignant and extremely resistant to treatment. The whole face may be on the verge of malignant change due to overexposure, and any attempt at treatment will be futile, as new growth may spring up at the margin.

Note.—The author wishes to thank Dr. E. M. Medlar, associate pathologist, University of Wisconsin, for valuable suggestions in the discussion of the pathology of prickle cell carcinoma.

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Carcinoma spinocellulare of the external nose following prolonged exposure to the X-rays. The section shows an abundance of the acanthous or prickle cells.

XCIV.

TRANSLUCENT NORMAL EAR DRUMS.

BY MYRON METZENBAUM, M. D., F. A. C. S.,

CLEVELAND.

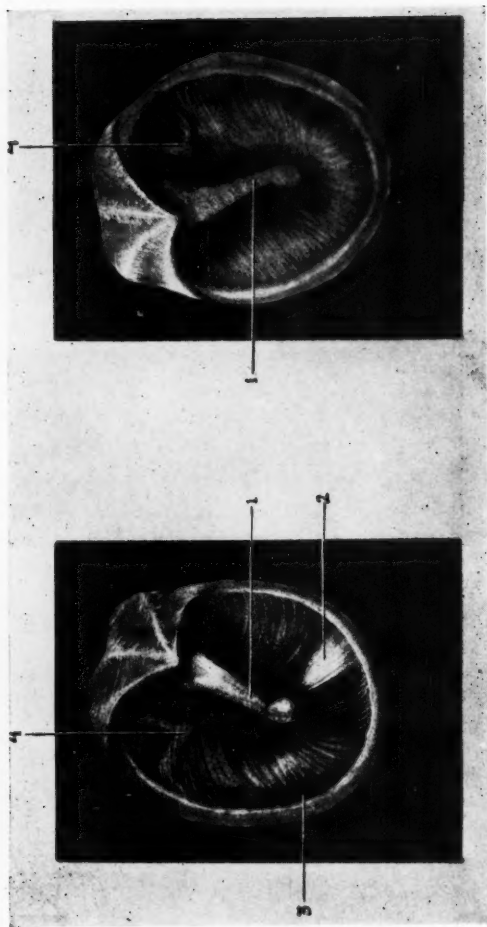
On inspecting a normal ear drum, the following details may be seen: The short and long process of the hammer, the umbo, the triangular light reflex and the anterior and posterior folds.

In rare instances the drum membrane may be so translucent as to reveal the incudostapedial joint, the niche of the round window and ostium of the eustachian tube.

The accompanying drawings are those of a woman, aged 38, who consulted me about her throat, and in the routine examination both ear drums were found to be so translucent that in addition to the usual details the incudostapedial joint could be clearly seen through both ear drums, and the niche to the round window could be seen through the right drum.

The functional ear tests were all normal.

736 ROSE BUILDING.



1. Hammer handle. 2. Triangular light reflex. 3. Niche of the round window.
4. Incudostapedial joint.

ILLUSTRATION OF DR. MOSHER'S PAPER ON
TEACHING METHODS.*

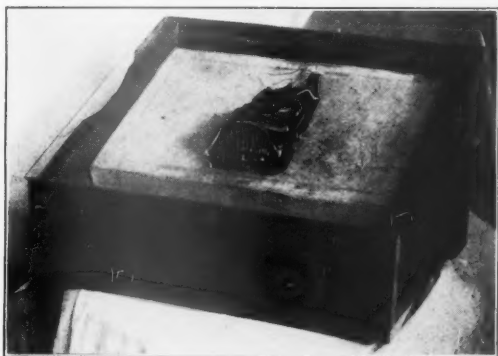


Fig. 1.

Fig. 1 shows the tracing box. With this it is easy to get an accurate tracing on the glass front of any anatomical specimen placed in the adjustable cloth tray. The tracing is made with a grease pencil. It is transferred to paper and the drawing finished free hand.

The tracing box makes it possible for the student to obtain a drawing of real value with little effort. I should never have the courage to ask postgraduate students to make a full series of drawings of representative anatomic specimens illustrating the applied anatomy of the nose and throat were it not for the tracing box.

*See page 721.



Fig. 2.

Fig. 2 shows the head holding frame. It is adjustable and has a dripping board and pan.

Beginners in laryngology practice the various laryngologic examinations on a wet specimen inserted in the frame.

The frame can be used for holding specimens during the performance of many intranasal operations.



Fig. 3.

Fig. 3 shows the method of practicing the examination of the larynx.

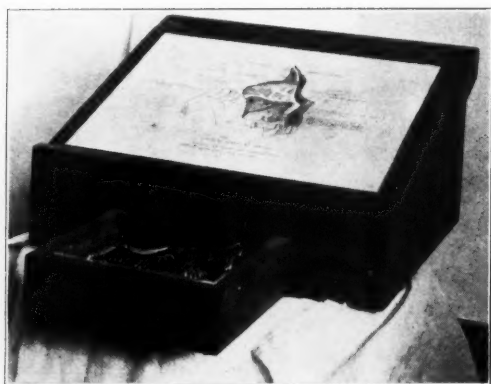


Fig. 4.

Fig. 4 shows a box for holding an annotated specimen. The anatomical specimen is kept in the tray attached to the front of the box. On the face of the box there is a glass covered annotated drawing of the specimen.

The student can handle the specimen as much as he likes. The drawing identifies for him the various anatomical structures. Beginners in laryngology find these annotated specimens very helpful.

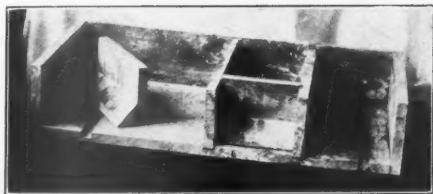


Fig. 5.

Fig. 5 shows the box used for pouring plaster casts. Three different widths of box can be obtained by moving the partitions. The partitions fit into grooves and so can be easily changed. The length of each box can be varied by moving the adjustable end.

The figure shows the box set for the second size. The movable end for the third or largest size is shown to the left.

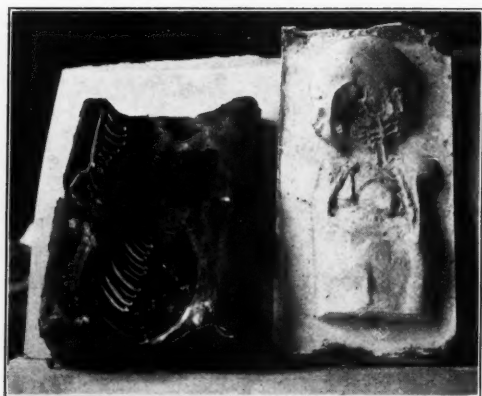


Fig. 6.

Fig. 6 shows on the left a glue mould, and on the right a rough cast taken from such a mould. The mould was made from a baby dissected to show the blood supply of the tonsil.

All the casts are natural size. The illustrations—this one and those which follow—are so reduced in size that most of the detail is lost. The illustrations, therefore, are of no anatomic value. They do, however, show the possibilities of casting anatomical specimens.

One of last year's postgraduate students told me recently at a medical meeting in Chicago that he valued the casts which he made in the February-March Course more than anything else. He keeps them about for ready reference.



Fig. 7.

Fig. 7 shows two casts by Dr. Ernlund. The upper cast shows the outer wall of the nasal fossa, the lower cast the antrum with the operative opening into the lower meatus of the nose.

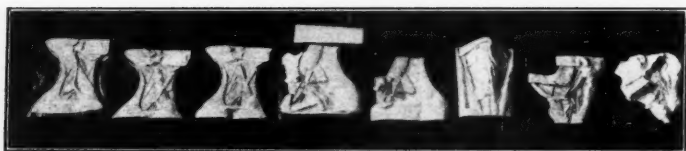


Fig. 8.

Fig. 8 shows a series of eight casts of a progressive dissection of the side and front of the neck. Dr. Hoople.



Fig. 9.

Fig. 9 shows a series of four casts giving the relationships of the thymus gland at birth. Dr. De Cloedt.



Fig. 10.

Fig. 10 shows three casts illustrating the blood supply of tonsil. Center cast—Dr. Kao—shows the arterial supply of the neck and tonsil. The two smaller casts—Dr. De Cloedt—shows the venous supply of the tonsil.

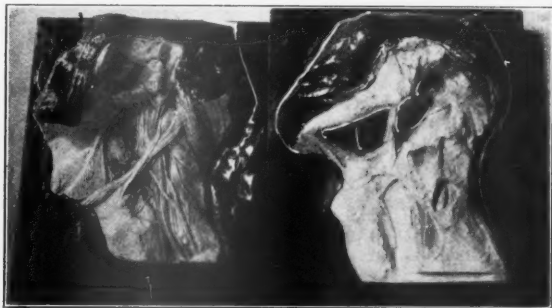


Fig. 11.

Fig. 11 shows two casts of dissections of the neck. Dr. Hoople.



Fig. 12.

Fig. 12 shows a part of the writer's collection of casts illustrating the anatomy and pathology of the esophagus.

The writer has had the assistance of many men in making the casts which compose the collection—notably Dr. Berry, Dr. Cahill, Dr. Wiggin, Dr. Harkins and Dr. Kirby.

OTOLARYNGOLOGY IN RELATION TO GENERAL
MEDICINE WITH A SPECIAL REPORT OF CASES
OF SCURVY AND BRONCHIECTASIS.

BY BURT R. SHURLY, M. D.,

DETROIT.

The modern trend of the highly developed differentiation in specialism has produced a finer line of demarcation between the general surgeon, the internist and the public health specialist. Medical education has been standardized so elaborately that its departments and heads, usually twenty in number, led to great difficulties in practical teaching and management. Borderline surgery and medicine should not become matters of rule, but should be governed by scientific qualification, choice and interest.

There is danger in the development of otolaryngology to an ultra-specialty that ignores the study of systemic disease. It must not be forgotten that many conditions of the upper respiratory tract are wholly systemic in origin and must be treated on a broad and liberal basis.

The Association of Medical Colleges has issued an edict that the faculty shall be grouped under four divisions—Surgery, Medicine, Public Health, Gynecology and Obstetrics—and all departments be merged under these divisions.

The close interrelationship of pulmonary abscess and tonsillectomy (Hedblom reports 54 such cases operated), of laryngeal tuberculosis and the pulmonary variety of hyperthyroidism and tonsillar ring infection, of mediastinal disease and laryngeal paralysis, of tonsillar infection and rheumatism, of psychoneuroses and focal infection, call for renewed interest in general medical diagnosis.

With the passing of the general practitioner, with the increased intelligence of the patient, with the diminution of referred cases, there must come to the specialist a greater interest in general medical diagnosis and therapeutics.

Along this line of thought I beg to submit the following five cases, associated with general and medicolegal medicine, for your consideration.

G. K., age 28; adult male; well nourished; admitted to the Detroit Eye, Ear, Nose and Throat Hospital January 3, 1925, with the following history: Father died of cerebral hemorrhage; mother living and well; three brothers and two sisters; **no history of hemophilia**; wife and daughter living and well. Past history: Diphtheria at four years, with good recovery; rheumatism at sixteen years without complications. Had frequent colds and sore throat until 1919, when tonsillectomy completely relieved this tendency. Three years ago patient suffered from abdominal pain and distention, with constipation. Nine months ago he consulted a surgeon, who diagnosed appendicitis and advised a rigid diet free from fresh vegetables and fresh fruits. This typewritten diet, consisting of five meals per day, was followed absolutely during a period of nine months. When this diet was ordered by a surgeon, March 24, 1924, the blood count was hemoglobin 80 per cent, red cells 4,810,000, white cells 9,200, lymphocytes 7 per cent; urine negative. On January 3, 1925, the blood count was hemoglobin 40 per cent, red 1,840,000, white cells 7,350, index 1.1, differential polys 61, lymphocytes 32 per cent, transitional 4 per cent, eosinophiles 2 per cent, basophiles 1 per cent; non-nucleated or stippled red blood cells; coagulation began at nine minutes; fragile at fifteen minutes and very little firmer at twenty minutes. X-ray showed no bone involvement. On this date, January 3, the patient entered the Detroit Eye, Ear, Nose and Throat Hospital as a stretcher case with the following symptoms and findings:

Marked hemorrhage from time to time from ears, nose and throat; bleeding gums; pain and swelling right knee, later left knee, followed by pain in the lumbar region—too painful to sit up. Hemorrhagic areas over right knee and internal aspect of both legs and thighs; urine highly colored, but no albumin or sugar found—specific gravity 1.026. X-ray and clinical examination of chest negative; basal metabolism 13.24 per cent, or 44.12, with 39.5 as normal; blood pressure 132; calcium content from estimation on blood plasma 10.18 with normal 9 to 11. Examination of the feces was negative. The alarming

blood picture, pending a diagnosis, was met with intravenous calcium and iron and a full diet of fruit and fresh vegetables.

We gave consideration to the following differential facts for diagnosis. Examination of the nose and throat and ears was negative. The temperature range was from 103 to 99. The question of differential diagnosis brought into consideration hemophilia, purpura hemorrhagica, pernicious anemia, anemia with parasites in the blood, relapsing fever, lymphatic anemia or scurvy.

Three outstanding features were negative—family history, no foreign residence and lack of sufficient red cell destruction or increased eosinophiles, ruled out hemophilia, relapsing fever and parasitic anemia, such as malaria or filariasis or trypanosomes.

It was not classified as splenomedullary leucemia, because there was no hypertrophy of the spleen or an increase in the number of leucocytes. It was not lymphatic anemia, because there was no lymphatic enlargement, large spleen, increased leucocytosis or small lymphocytes in the differential count. In pernicious anemia the color index is usually above one, and nucleated red cells may be seen. The blood picture, made three days later, following forced feeding of fresh vegetables, fruit juices, intravenous iron and calcium with Bland's pills, showed a striking improvement. Hemorrhages ceased, the edema in the tissues rapidly subsided, joint and muscle pains faded. The blood report on January 6th (three days later) was reds 3,496,000, leucocytes 5,100, hemoglobin 60, color index .85, polynuclears .75, lymphocytes 14, large mononuclears 2, transitionals 6, eosinophiles 1. Coagulation time, $5\frac{1}{2}$ minutes; firm clot, 9 minutes. The patient steadily and rapidly improved and on January 9th the blood count was reds 4,420,000, leucocytes 6,500, hemoglobin 70, color index .9, polymorphonuclears 64, lymphocytes 31, transitionals 3, eosinophiles 1, basophiles 1. The red cells show a moderate variation in size, shape and staining quality. No stippled or nucleated red cells were seen. Coagulation time, five minutes, with a firm clot in eight minutes. Pain had subsided entirely. No fresh hemorrhage occurred, and the purpuric areas had faded. The patient was discharged from the hospital in eight days on full house diet with an excess of fruit juices.

A complete count, January 13th, showed red cells 4,080,000, leucocytes 7,350, hemoglobin 80, color index 1, polymorphonuclears 62, lymphocytes 27, large mononuclears 2, transitionals 7, eosinophiles 1, basophiles 1; coagulation time, 4 minutes, firm clot in five minutes. This case exhibited the factors of faulty diet, weakness, mental depression, hemorrhage from the gums, ears and nose, with purpura and subperiosteal hemorrhage and tenderness. The diagnosis was confirmed by the series of blood counts and the rapid recovery with iron and calcium intravenously and by mouth, together with an abundant diet of meat, fresh fruits and vegetables. The anti-scorbutic vitamin water soluble C, found particularly in oranges, lemons, limes and tomatoes, with vitamin D, in red bone marrow, with a tonic rich in calcium and containing malt, pulverized ossis, lactic acid, pancreatin and pepsin, were continued for some months. At the present time the patient is well.

Case 2.—Bronchiectasis, while strictly belonging to the internist, becomes of otolaryngologic interest from the fact that many cases are primarily sinus infection, and that three distinct measures for relief fall directly into our special field. The condition is frequently overlooked clinically. Eighty-four per cent of the cases, according to Moore, may be definitely diagnosed with the X-ray. In my own cases the picture of dilated tubes and saccules filled with pus over the lower lobes could be seen to empty with cough and expectoration. Moore distinguishes three varieties, the infiltratic, the cylindrical and the sacculated. The characteristic profuse sputum, without tubercle bacilli, the violent paroxysmal morning cough, the offensive odor, the physical signs of cavernous breathing, bronchophony and whispering pectoriloquy with coarse and metabolic râles, temperature 99 to 100, give suggestive evidence confirmed by the fluoroscope.

These symptoms and findings were present in Mrs. C., age 22, housewife, who consulted me October 6, 1924. The previous physician diagnosed pulmonary tuberculosis. Following tonsillectomy, one and one-half years ago, the patient lost weight. Cough and expectoration increased and temperature developed. Physical and X-ray examination: The hilus density markedly increased and contains numerous dilated bronchi.

Both bases show an irregular diffuse infiltration through which are evident the honeycomblike structures, probably the result of dilated bronchi. The apices are clear and the lung fields clear with the exception noted. Frequent sputum examinations showed staphylococcus, pneumococcus and streptococcus. Examination of nose and throat showed bilateral antrum infection.

In this type of case, otolaryngology has three therapeutic aids to offer over that of the internist—that is, puncture, irrigation and drainage of the sinuses, intratracheal medication with guaiacol and camphor to facilitate drainage of the bronchial tubes and bronchial lavage with the proper bronchoscopic tubes and irrigating apparatus. Under this procedure the patient obtained great relief. The temperature returned to normal, the cough was greatly improved and odor subsided. Irrigation has been continued at intervals until the present time, although the patient is seven months pregnant.

The interrelation of medicolegal medicine and otolaryngology has become more intimate since the war, and report of three cases of personal experience may prove of interest. During the last few years one in every hundred doctors in Michigan has been sued for malpractice.

Case 1.—C. T., a girl of 21 years, was under the care of Dr. H. The charge in this case was that in the removal of the plaintiff's tonsils the doctor improperly and negligently attempted to remove the tonsils, and in so doing removed the uvula, the palate and anterior pillars in the plaintiff's throat, cut and tore the walls of the pharynx and the muscles of the throat, which resulted in a complete atresia of the nasopharynx. The plaintiff had formerly (according to her statement) a splendid voice, and as a result of this injury she was incapacitated from carrying on a musical education. The patient, after commencing suit, came to the Detroit Eye, Ear, Nose and Throat Hospital with a complete atresia of the nasopharynx, traumatic in origin. I operated and secured an opening an inch in diameter. A dentist manufactured a holding device that was attached to the molar teeth. This was imperfect, however. Scar tissue formed, and after seven years the contraction has been so great that the nasopharyngeal opening will admit only a probe the caliber of a lead pencil.

At the trial there was no question about the damages, but in order for the plaintiff to have her case submitted to a jury it was necessary to procure some medical witness familiar with the practice in the community or similar communities in which Dr. H. practiced to testify that in the performance of the operation the physician used some method not in accordance with the usual, ordinary practice in that community, and that as a result of his failure to use that ordinary method the damage probably followed (it is safe to say that it probably followed), or that he omitted to do something which was ordinarily done by physicians practicing in that and similar communities, and that as a result of the omission the damage probably resulted. It was the theory of the defense that some blood condition existed, and, while the charge was not openly made, plaintiff took it up as an open challenge and a Wassermann test resulted in negative findings.

At the first trial the case was submitted to the jury on the testimony of an X-ray specialist, who formerly had been in general practice, regarding the faulty position in which plaintiff was placed at the time of the operation. The jury disagreed, even though the physician had offered to pay \$6,000.00 in settlement.

At the first trial I refused to appear, on account of family sickness, and, as the attorney for the plaintiff was unable to subpoena me, it was agreed that a written statement would be admitted.

At the second trial a subpoena was served upon me and I was forced to appear. I was on the stand for an entire day. The attorney for the plaintiff tried to get a statement from me which would carry the case to a jury, and it looked as if the judge was going to direct a verdict of no cause for action, but he felt so sorry for the girl that he asked Mr. Barbour, attorney for the defendant, to try to get the doctor to pay something, and that he would endeavor to get the plaintiff's father, the plaintiff and the attorneys for the plaintiff to accept it, and eventually \$1,000 was paid to the girl, \$250 to the attorneys and the doctors' bills were paid, and the case was settled.

The second case was C. F. vs. Dr. J. P. T. Dr. T. was more of a physician than surgeon, but had considerable ex-

perience in the removal of tonsils. He was called upon, in the course of his practice, to remove the tonsils of the plaintiff. In removing them he left a portion of one tonsil, and the plaintiff claimed she continued to suffer from neuritis and rheumatism as she suffered before the operation, until I removed the remaining portion of the tonsil, some months afterwards, at the request of Dr. T., when she became perfectly well.

There is no doubt that Dr. T. left a considerable portion of both tonsils. I was called as a witness by the attorney for the plaintiff to establish negligence. On being questioned, it was stated that, in my opinion, the average physician might easily remove a portion of the tonsil, and even a specialist might do so if the hemorrhage was especially great, and that it might be better to remove the remaining portion at a later time. It was stated that if I were sued for all the pieces of tonsil I had left in the last thirty years the Wayne Circuit Court would have little else to do. Every effort was made to call out statements that the average practitioner in this community would be guilty of negligence if he left a portion of the tonsil and that such portion of the tonsil would probably cause a continuance of the neuritis and rheumatism. The court directed a verdict of no cause of action in favor of the physician.

The next case to which I wish to call your attention, in which I was the defendant, may not be considered as strictly falling within the domain of otolaryngology, since it was a case of death occurring during the administration of an anesthetic; we never proceeded as far as the tonsillectomy itself. Inasmuch, however, as the administration of the anesthetic may be correctly termed a necessary part of the operation, and since some useful information may be derived from my experience in this case, I have decided to bring it to your attention.

In August, 1922, Mrs. E. B., widow, in company with her woman friend, who claimed to be a practical nurse, consulted me with reference to performing a tonsillectomy upon her son and removing his adenoids. The son did not accompany her. He was, physically, a fine appearing young fellow, nineteen years old, six feet tall and weighed 175 pounds. According to her statement, he had been attending a military institute

in Ohio, had been suffering from tonsillitis and had been advised by the resident physician of the institute to have his adenoids and tonsils removed. An appointment was made for him for the following morning, and he appeared with the nurse at the appointed hour and was examined. In accordance with our general routine, where not otherwise arranged for, preparations had been made for a general anesthetic.

He was preceded upon the operating table by a woman patient from Morenci, Michigan, to whom a half of one per cent solution of procain was administered and who, as she was leaving the operating room, said in the presence and hearing of the young man (who was outside the operating room awaiting his turn) that she was feeling fine and that her operation had been a splendid success.

He entered the operating room in my absence, my anesthetist and a surgical nurse being the only other persons present at the time. Upon entering the room, he inquired what anesthetic the preceding patient had had, and was informed that it was a local anesthetic; whereupon he asked why he couldn't have the same thing and was told that he could. He then expressed his desire to have the same anesthetic administered to him that the preceding patient had received.

In accordance with this request, the anesthetist, who is a graduate surgeon, proceeded to administer the same solution of procain and had injected two drams into the posterior pillar when the patient suddenly collapsed and, after about an hour spent in unavailing efforts to revive him, died.

The mother was sent for, and, of course, asked how the death had occurred. Relying upon the information received from the anesthetist and surgical nurse, I informed her that while *procain* was being administered as the anesthetic he had collapsed and died.

Some time thereafter I was sued for malpractice by the mother, as the boy's administratrix, she claiming \$50,000 in the name of his estate.

She had employed a firm of very skillful lawyers who, I am informed by my counsel, departed from the usual practice of attorneys in malpractice cases and, instead of suing, as the lawyers term it, "in tort," or, in common language, for the

negligence or malpractice of the physician, claimed that she had told me of having been warned by her family physician (conveniently dead) never, under any circumstances, to permit cocain to be administered to him. She claimed that I had agreed not to administer cocain or any of its derivatives; that I had administered cocain, or one of its derivatives, and that I had told her that I forgot to tell the anesthetist to administer a general anesthetic, and not cocain or other local anesthetic, and that the cocain had caused his death; and, instead of claiming, as is usual, damages for the negligence, she claimed damages for this claimed breach of contract.

My counsel informed me that this was a very cunning device, inasmuch as it might cut off my right to demand that she prove the malpractice by the testimony of surgeons, as is the rule of law in ordinary malpractice cases, and that it would leave the case to be presented to the jury, on this phase of the case, merely upon the testimony of lay witnesses, whom the jury might believe as against my uncorroborated denial of the fact that any such agreement had been made by me.

Counsel said, however, that this would still leave the case in such condition that plaintiff must produce such medical testimony to the effect that the death did really result from the administration of cocain, and which would warrant the jury in finding this to have been the fact, and that if she did not present such testimony, we would still have the right to demand that the judge direct a verdict in our favor because of failure to prove the contention.

It will be quite apparent to you, I think, that, as it was claimed by my lawyers, plaintiff's claim was founded upon the similarity of the two words "*procain*" and "*cocain*." I did, in fact, tell her that "*procain*" had been administered; but not being familiar with the name of the drug, she probably understood me to say "*cocain*," and she must have stated this to her lawyers.

There was no medical testimony produced by plaintiff tending to prove that the boy's death was, in fact, caused by the administration of cocain, and none that cocain was, in fact, administered, but the able judge who presided at the trial held that sufficient testimony had been developed by her own and

her companion's testimony that I had admitted the use of cocaine (which was untrue), together with other testimony, to warrant submitting the case to the jury.

The trial occupied a week, and the jury promptly returned a unanimous verdict in my favor.

One very interesting point of law was developed when the trial judge held that the boy, although still a minor and under his mother's guardianship, had arrived at such a stage of mental development as to give him the right to choose what anesthetic should be administered to him, and the case was submitted to the jury upon that basis.

Under all the circumstances, it was very clear that this was a case of thymic death, and my associate, Dr. Gaines, and Drs. J. B. Kennedy and Frank T. F. Stephenson, as well as I, testified to that effect in response to hypothetical questions covering the proven facts of the case. No postmortem was held. The coroner gave the cause of death as paralysis of the phrenic nerve. No one knows the real cause of death, however, which is greatly to my advantage legally.

Aside from the medical and surgical facts involved here, my counsel have made these suggestions as to the legal aspects of the case:

1. In all your cases be very careful that at least one physician besides yourself is present at the interview where arrangements for an operation are made.

2. Wherever possible, have the details in writing. This can be done in the manner of taking a history, jotting down the points in the presence of the person or persons with whom the arrangements are made, and finishing the memorandum with a full statement of the details of the anesthetic, operation, perhaps the price, or confirming the arrangement by letter in some other way.

I should say that this case is not yet finally settled legally, since the plaintiff has appealed to the Supreme Court from the judgment in my favor.

Under the law, as I am informed by my counsel, no new testimony is to be taken, and the verdict of the jury and the judgment based upon it must stand unless it can be successfully demonstrated to the Supreme Court that the trial judge committed some substantial, legal, prejudicial error, either in

the admission or exclusion of evidence, or the substance of the instructions, or charge, which he gave to the jury.

In my opinion, and that of my counsel, this case was what is called a "frame up." One of the proofs that it was such is apparent in the claim that the word "cocain" was used. For ten years "procain" has been so commonly used as my local anesthetic in tonsillectomies that it has become a matter of routine with me and my assistants, and I could not, therefore, have used the word "cocain."

Another proof that it was a "frame up" is furnished by the fact that, about a year after the boy's death and while the suit against me was pending, I received word that an out of town physician had agreed to hold an autopsy upon the body. My counsel and I and several other surgeons were present at the time and place secretly arranged for the autopsy. We waited a long time before anyone else appeared. Finally, someone did appear who, immediately upon seeing us there, backed out of the room, waited on the outside for his companions to appear, held a whispered conversation with them and apparently communicated with the plaintiff.

After we had waited nearly four hours, the undertaker said that the mother had telephoned, and was then on the telephone, and had given strict orders that no autopsy should be held so long as I was present. This statement was confirmed to me over the telephone by the mother herself.

This experience serves to emphasize the great care necessary in taking all possible precautions in dealing with strangers.

XCVI.

INSULIN IN DIABETES WITH MASTOIDITIS.*

By THOMAS C. GALLOWAY, M. D.,

EVANSTON.

Even before the discovery of insulin, there were developing new ideas concerning operative relief in diabetes mellitus, but with this powerful new weapon the whole attitude toward its surgery has changed. From a view almost despairful in the presence of an infection which demanded intervention, now has come considerable assurance in the face of such complication. As Allen says, we have reversed the old rule, which was "to avoid as far as possible all surgery," and now the importance is quite generally recognized of removing the overload which infection imposes on the diabetic organism.

A voluminous literature has appeared since the first papers on insulin, but there is little mention of its bearing on otology, and it seems worth while to report an unusual case of mastoiditis in a diabetic with only doubtful glycosuria, the recovery of which was made possible by insulin, and to discuss the principles of treatment.

Case Report.—S. B., a 53-year-old widow, a cook, following a cold, had a spontaneous perforation of the right drum membrane after an earache of one day. When first seen, eleven days later, she looked rather toxic and complained of marked persistent pain around the right ear. Profuse serosanguinous discharge containing streptococci was present, the posterior superior canal wall drooped, there was marked tenderness over the mastoid tip and moderate swelling over the mastoid. The white blood count was 15,600 and the temperature 100 F. During the following two days the pain persisted, and the general condition became worse, although the temperature and the white blood count did not rise. Thirteen days after perforation of the drum I did a simple mastoid operation under gas-oxygen anesthesia, finding a small mastoid, moderately

*Entrance thesis read before the Chicago Laryngological and Otological Society, April 6, 1925.

broken down, filled with pus and granulated debris in tip and deep cells, and a forward lying sinus which was uncovered over an area 3 mm. wide. The temperature rose to 101.8° F. after the operation but fell at once to 100° F. Cultures taken from the mastoid at operation showed streptococcus hemolyticus.

On the third day after operation there was a pale, doughy swelling around the mastoid wound. The next day this was pink, and by the fifth postoperative day presented the typical raised, red, tender, sharply circumscribed swelling of an erysipelas. This went into the scalp and nearly to the shoulder, but did not later extend beyond the cellodion painted borders. By the eighth postoperative day it had receded to the wound margins. The wound showed no attempt at repair at this time. The patient complained of pain in the vertex and right ear and was sleepless and restless. The temperature was below 100 F. until the tenth day, when it rose to 101 F., dropping down again to 100 F. The symptoms had been ascribed to meningeal irritation, but on the eighteenth postoperative day I noted that reflexes, fields and eyegrounds were normal, with no rigidity of the neck.

At this time the skin margins were just as they were three days after operation, but the periosteum had freely sloughed out, as had the sternomastoid tendon fibers, and the bone was widely denuded. There were no granulations present. Up to this time I had ascribed the lack of wound reparation to the damage done by the erysipelas, but rather late awoke to the fact that some other explanation must be found.

The blood Wassermann was negative.

The urine on admission gave a doubtful Haines' test for sugar, but the next specimen was negative, as was one the following day. This indicated that no important degree of diabetes mellitus was present, but rather in desperation a blood sugar determination was ordered, which showed .308 per cent. The urine then showed considerable amount of acetone, not previously sought in the routine examination. The respiratory curve was again surveyed, and the high rate, previously dismissed as due to a bronchitis, with the headache and dyspnea, suggested that the patient for some time had had a high degree of acidosis, though the blood plasma CO_2 when taken two days later, was 53.8 per cent, within normal range. A

blood culture taken at this time showed the same type of slowly hemolyzing streptococcus recovered from the mastoid at operation, the urine constantly showed albumin and casts, and the prognosis seemed absolutely bad. Dr. Rufus Stolp was called in and instituted careful dietary regime, with fairly small doses of insulin, never more than ten units daily, with the result that the blood sugar soon approached normal, and the patient rapidly recovered from her headaches, restlessness and dyspnea. By the twenty-second day after operation, when the blood sugar was down to .216 per cent, the first definite granulation appeared. From that time improvement was steady, but slow, as might be expected from the condition the wound had reached. At the end of two months after operation the patient had a dry ear and a healed wound.

Comment.—In retrospect, it seems clear that the diabetes should have been diagnosed early, and energetically treated from the outset. The erysipelas was too readily accepted as the cause of poor healing, and the doubtful finding of sugar in the urine dismissed when subsequent specimens were negative. Joslin believes that any case which shows glycosuria at any time, except after very high carbohydrate ingestion, should be regarded with extreme suspicion, and Reveno reported a case in pregnancy which showed no urinary sugar by the ordinary tests, though the diabetes had gone on to coma. His case, as did this one, had nephritis, which may be accepted as an explanation of the very high kidney threshold.

It may well be, however, that this patient was not a true diabetic in the beginning but that the infections precipitated the disease. The gratifying thing was that once the real condition was recognized, though it seemed desperate, a proper regime with insulin soon brought it under control.

Discussion.—Aside from any etiologic relationship of focal infections, it has come to be recognized that sepsis plays a most important part in established or potential diabetes. By it a partial diabetes may become complete, carbohydrate tolerance painfully established may be quickly lost, a defective pancreas which, under regulation, has been fairly adequate, may suffer further irreparable damage, and death from infection or coma quickly supervene. Frissell recently reported that in six cases of coma, five of these were precipitated by infection.

Diabetics, as is well known, are very prone to infections, and the infections themselves have a most adverse effect on the disease, and thus there is produced a true vicious cycle.

The procedures for infections in diabetes seem to have become very well agreed upon. In the first place, proper medical care is essential, and the advances made in the management of diabetes and the involved questions of dietary balances require the expert direction of an internist with the aid of laboratory control. Without going into detail it may be stated that in an ordinary case of uncomplicated diabetes mellitus, a maintenance diet with a condition of slight undernourishment is desirable, that sufficient protein should be taken to replace body waste, that carbohydrates should be given in amounts not too great to overload the impaired pancreas and yet sufficient to carry on the combustion of the fats, which is essential to prevent acidosis. Careful dietary regulation, it is emphasized, is the basis for all diabetic treatment, and no carelessness in this is to be covered up by the use of insulin. This most valuable and frequently life saving agent is to be reserved for the cases in which proper nutrition cannot be maintained without its aid, and for the complications—sepsis, acidosis or coma. While in uncomplicated cases a short period of observation of the patient's response to his usual diet or a test diet may be desirable to determine the need and dosage of insulin, in cases with infection insulin should usually be given at once, in dosage regulated by the per cent of blood sugar or urinary sugar, with carbohydrate added if necessary to prevent a condition of hypoglycemia, and fats should be sharply limited or withheld completely.

Since infection is a most potent cause for harm in diabetes and predisposes to sudden change for the worse or to coma, if such infection is amenable to surgical treatment, operation should not only be done but should be done earlier than in the corresponding situation in the nondiabetic. For otology we may draw parallels from the wider experience of general surgery. For example, according to Joslin, one should not wait for a carbuncle to localize. Only if the local advance of the infection seems definitely arrested and the general condition of the patient satisfactory may delay be permitted. With gangrene of an extremity, dry and forming a definite line of

demarcation, and blood sugar relatively low and general condition good, one may wait. If the gangrene is moist and progressive, or the general condition not good, with carbohydrate tolerance markedly lessened, or markedly increased amount of insulin required for its utilization, and acetone bodies present in the urine, haste is imperative.

No rule can be formulated for mastoid surgery in diabetics. It would seem, though, that the usual time for interference must be shortened, as the limiting processes may be diminished or absent and a localization in an excavated mastoid is not usually attainable. If, however, there are not signs of retention or sepsis in a suppurative otitis, the presence of bone destruction is doubtful and the diabetes itself stationary or improving, especially if the diabetes had been previously unknown and the case had no previous benefit of dietary regime and insulin, it would be well to wait. It may be that in such a case improvement in the diabetes may so better resistance that the infectious process is conquered. Joslin has noted repeatedly that where pus pockets were present around teeth, treating the patient to withstand extraction so built up resistance to infection that intervention was unnecessary. A second ear case seen recently, with profuse discharge, some tenderness over the mastoid tip on the tenth day and high urinary sugar and acetone might have seemed an advisable case for operative interference, but there was no temperature, the patient's general condition was improving and relatively simple dietary regulation made the otitis clear up.

For preoperative preparation, if no emergency exists, a diabetic regime for a few days is desirable, using insulin under careful laboratory control, in relatively large doses to hasten the improvement, such doses being buffered with proper amounts of carbohydrate to prevent the development of hypoglycemia. Fluids to flush out the acetone bodies, and food, especially carbohydrates, to combat the acidosis should be given as late before the operation as possible, and the period between preoperative and postoperative alimentation should be short.

Ether, in diabetics, for this reason and also because it diminishes urinary excretion, upsets the stomach and has a possible detrimental effect on the pancreas itself, is usually not advisa-

ble as an anesthetic. Many bad effects have been reported in diabetes with it, but the Mayo Clinic, by short anesthetics, with small amounts of the drug and rapid, nontraumatizing operations secured excellent results. Chloroform should be taboo. Nitrous oxide oxygen, or possibly ethylene, would seem the preference for general anesthesia, but the best is local when possible. Premedication with morphin should be limited, because it depresses respiration and therefore the output of carbon dioxid.

It is proper here to point out that besides caring for sources of acute sepsis, chronic foci should be eliminated in the treatment of diabetes. The work of Banting, Allen, Harris and others indicates that pancreatitis, which many authors believe the precursor of diabetes, may be caused by certain abdominal conditions, notably gall bladder disease and intestinal infections, or such a pancreatic injury may be secondary to more distant foci, as carious teeth with abscesses and diseased tonsils. These should receive appropriate attention. The weight chart (Fig. 1) of a 13-year-old diabetic (G. H.) shows the remarkable improvement brought about with no change in diet or insulin dosage, by the removal under local anesthesia of badly infected tonsils.

It is interesting in this connection to consider the change in mortality in operations in diabetes. The improvement had already begun before insulin, but has been much greater since.

A surgical mortality of 30 per cent was found by Fitz at the Massachusetts General Hospital before 1918 in diabetics. Strouse, at Michael Reese in 1916, found a mortality of 31 per cent, though no deaths occurred in eight cases suitably prepared. At the Mayo Clinic, in 1915, the operative mortality of diabetics was 8 per cent; in 1921, 6.4 per cent. During the last two years Wilder and Adams, at the Mayo Clinic, have been able to report that with dietary control and the proper administration of insulin in 237 operations the mortality has been only 1.2 per cent by operation and 1.6 per cent by case.

It should be borne in mind, however, that surgery in diabetes may always become a serious matter when there is possible infection of a wound or when there may be induced severe disturbance of nutrition, and no promiscuous surgery is warranted in this disease. As Hempstead states, no surgery that is purely optional should be done. The certainly graver dan-

gers of operation in such cases must be balanced against the direct and prophylactic value of removing the infectious foci. Altogether, however, with the right medical management and insulin in reserve, proper surgical intervention may be undertaken in diabetes mellitus with relative security, and should be undertaken when it will remove an overload which is causing the breakdown of a defective organ.

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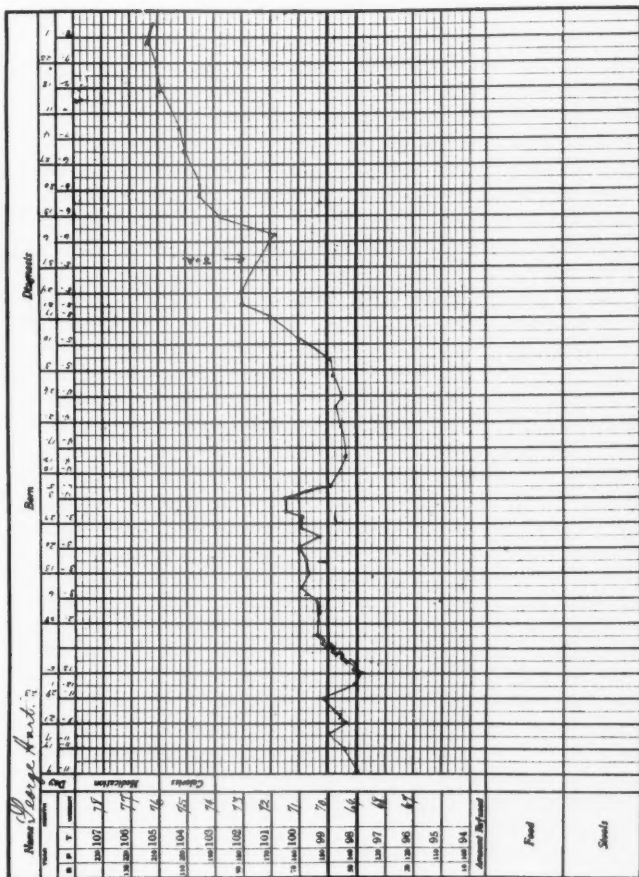


Fig. 1.

XCVII.

REPEATED CORYZAS. PROPHYLAXIS AND TREATMENT.

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Economically a cold in the head is a costly disease, when we take into account the slowing up of sufferers who are trying to do their usual work, and those who give up work entirely for a few days. To those who recover by themselves, we must add those who have prolonged convalescence under the belated care of a physician. Because so many colds are self limited, and because the measures of value in shortening the duration, or preventing colds, cannot be stated in a few definite terms, we have allowed our patients to believe we are helpless in these everyday affairs, and, too often, we discourage them from seeking our advice, even when we know that repeated coryzas mean either that some chronic trouble is already present or some sinus or ear complication quite likely to follow. As to what can be done in these conditions, I should like to add my limited experience.

Regarding the etiology of coryza, there is still some discussion. Cecil examined a number of cases of coryza and found some bacteria free, more than one-half with streptococcus viridans predominating, others with pneumococcus and streptococcus hemolyticus. He was inclined to believe that most people suffer from one particular organism which predominates for them.

Mathers, in his investigation, found the streptococcus hemolyticus predominating in the majority of cases and believed that the particular variety of cold which he was investigating was due to that organism.

Foster took the nasal discharge from typical coryza cases, passed it through a small Berkfield filter, and reproduced the coryza from the dilution of the filtrate, wherefrom he concluded that common colds were caused by a filterable virus.

Eaton emphasizes the fact that the common cold is an infection and not merely a congestion.

Sawtelle asserts that chronic focal infection registers its effect upon the sensitive nasal tissues, and has seen marked improvement from the drainage of a gall bladder, the removal of an appendix, the correction of a paranasal sinus disease, the relief of gastrointestinal and genitourinary disturbances, the removal of infected tonsils and teeth, and finds simple chronic sinusitis to be often the reflection of a protein poison.

Mudd, Grant and Goldman have a very exhaustive paper on the etiology of acute inflammation of the nose, pharynx and tonsils, and conclude that:

1. The filterable virus of Kruse and Foster, inducing apparently a clinical entity, a type of acute coryza. According to the experiments of its discoverers, this is of relatively high virulence, and may cause infection practically independent of the action of exciting factors.

2. Various bacteria, including the pneumococcus, streptococci, *B. rhinitis*, *B. diphtheriæ*, Friedlander's bacillus, *B. influenzae*, and probably also *M. catarrhalis*, *B. septus*, *M. paratetragenus*, *S. aureus*, the meningococcus, Vincent's spirillum and fusiform bacillus, and possibly others seem to be capable of inducing infection of variable extent, duration and symptomatology. The relative virulence of the microorganisms also varies within wide limits, both between themselves and from time to time in the same organism, in some instances high, infections of epidemic proportions, largely independent of exciting factors, may be produced; in other instances low, sporadic infection may occur only when some factor or factors serve to depress resistance, general or local, to the point of vulnerability.

3. Protein sensitization, the basis of vasomotor rhinitis and of true bronchial asthma, is the underlying cause also of a relatively infrequent subgroup of acute recurrent "colds."

4. Various systemic diseases, drugs, mechanical, thermal and chemical trauma, chronic nasal affections and reflex neurones are causative.

One factor by which resistance to bacterial infection may be lowered is excessive chilling. Their experiments had to do with the effect of chilling the body as an excitant of infection.

They demonstrated that the mucous membrane became ischemic with vasoconstriction instead of vasodilation, as had been previously presumed. They rather favored Wright's suggestion that the local biochemical change which increases infection might not come through the sympathetic nervous system. They concluded that chilling the body surface lowered the resistance to bacterial infection. They also draw attention to Leonard Hill's monograph on the susceptibility of persons staying in an illy ventilated room and then going out into the cold atmosphere, and urge the importance of contagion and the effects of indoor life with lack of outdoor exercise.

Stoker believes that cold is due to an infection from some focus, as the tonsils, teeth, or accessory sinuses, when there is a lowered resistance or an inherent weak resistance to the causative organisms.

In contradistinction to most other writers, Ferreri believes that microorganisms in a cold are only secondary, arguing that if germs cause cold we should all have acquired immunity long ago. "We may have a thousand colds in a lifetime and still be susceptible." He believes the etiology lies most often in prolonged chilling of the surface of the body, to which those are more sensitive who have an irregular cutaneous vasomotor function. Acute rhinitis proper is due to a purely physical action of too low or too high temperature.

Gordon, in investigating the virulence of organisms, found that those present in a healthy nose and throat were just as virulent to mice and rabbits as those carried during an acute cold, and concluded that the micrococcus catarrhalis was but rarely the cause of a cold. Blassberg collected different opinions on the subject:

1. Bouchard explains that in pneumonia the effect of cold on the mucous membrane causes vasoconstriction, with a resulting disturbance in nutrition and damage to the epithelium, favoring the penetration of the cause of the disease.

2. Others are of the opinion that chilling causes an unfavorable distribution of the blood, resulting in catarrhal manifestations.

3. Lewkowica holds that chilling causes a purely mechanical injury to the epithelium, especially in persons with latent infections.

4. Catching cold is the result of disturbance in excretion and evaporation from the skin. This causes a retention of the injurious products of infection.

5. Rosenthal thinks that the chilling reaction is due to the arrival in the internal organs of the blood which is chilled at the periphery.

6. Chilling has a depressing effect upon the nerves of the skin, causing neurotrophic disturbances favoring infection.

7. Attention is also drawn to the incidents of colds in the various months, which are due to chilling. However, infection and intoxication, with other causes, are considered before chilling.

Max Gahwyler, after numerous experiments on animals, and reviews of the various theories, concludes that at present we can only say there are common colds. Of course, in this conception he leaves out seasonal and weather diseases and those from exposure, and believes three factors are important in the common cold:

1. A local sensitiveness to cold, or a predisposition.
2. Exposure.
3. Infection which supervenes on the damaged tissues.

Infection produces the disease from the local exposure in the same way, strictly speaking, that it produces a local or general wound disease from a tissue wound. Stricker considers that the predisposition exists in a lymphatic, arthritic and neuropathic constitution.

Other contributing factors, especially facultative pathogenesis of many bacteria, explain the great variation in the occurrence of common colds, not only in different individuals but in the same person.

Winholt and Jordan, in studying the epidemiology of colds in infants, found the majority occurred in families where some other member of the family, most apt to be the mother, had a cold.

Berry found that the most deadly of all causes of colds in children is contact with other members of the family who are infected. Malnutrition and loss of immunity are factors. Errors in personal hygiene, faulty environment and chronic condition, such as rickets, as well as nasal obstruction, are important.

Jordan, Norton and Sharpe, in a study of the common cold among students in California, Chicago and Galveston, found less difference in the groups than might be expected on the basis of climatic conditions alone. Nose and throat operations did not result in a marked reduction in the frequency of colds. Resistance building procedures apparently had little effect on the frequency. Twenty-two per cent stated they had possible contact with those having cold preceding their own. Sixty-four per cent believed their colds were induced by some strain on the heat regulating mechanism of the body. Bacteriologic investigation showed no special group of bacterium predominant. They concluded that outside influences, particularly the chilling of the body, may serve to induce a cold, even though it cannot be regarded as the most important etiologic factor.

Smiley, from four years' records of Cornell University students applying for medical advice for infections of the throat and respiratory system, found a gradual rise in numbers in the months of January, February and March, and a gradual fall as summer approaches. Through a questionnaire to the freshmen and sophomores, to determine the number of colds they might have without seeking advice, he found that 23 per cent had colds four or more times a year, 60 per cent two or three times, and 17 per cent once or not at all. They were unable to tabulate any major factors in the susceptibility from their information as to the personal habits, etc.

Robertson and Groves, in experimental tests with humans, with filtered nasal secretions from acute coryza, conclude that although the specific infectious nature of coryza has not been proved bacteriologically, there is overwhelming clinical and epidemiologic evidence of it. Climatic changes, exposure to cold and lowered resistance must be borne in mind.

Knowing the various etiologic factors advanced by others as the cause of colds, we quite naturally attempt a classification which fits in with our clinical experience, and which will help us in analyzing a particular case when it presents itself. Briefly we may classify the etiology of colds for a working basis as follows:

1. Filterable virus (Kruse & Foster), highly virulent and not influenced by other factors.
2. Various organisms.

- (a) In epidemics these may be so virulent as to need no other causative factor.
- (b) Where another factor, plus organisms, seems necessary to produce the disease.
- 3. Protein sensitizations—Pollens, foods, etc.
- 4. Focal infections.
 - (a) In nose and throat.
 - (b) In more remote locations.
- 5. Those coming with systemic diseases.
 - (a) Acute diseases.
 - (b) Chronic diseases (syphilis, tuberculosis, polyglandular).
 - (c) Nervous.
- 6. Occupational colds.
 - (a) Trauma, mechanical, thermal, chemical.
 - (b) Temperature, high or low.
 - (c) Weather.

To these we must consider the factors which, if not actually the cause, are highly contributory:

- (a) Exposure, chilling.
- (b) Lowered resistance, malnutrition.
- (c) Errors in personal hygiene.
- (d) Faulty environment, poor ventilation
- (e) Climatic conditions.
- (f) Obstructed nose.

With an outline like the above in mind, one is in a position to get a valuable history from his patient for the purpose of determining whether or not his particular condition can be helped. The numerous causative agents do not allow a simple or single line of procedure to prevent or shorten them, but, if a sufferer can be placed in a particular group, his colds may be very materially lessened. Repeated colds often cause extension to the paranasal sinuses or ears. In epidemics a single cold may cause extension, and depend largely on the organism predominating. In one epidemic the sequela will be sinus involvement, even a particular sinus predominating. At other times the sinuses escape and acute infection of the ears supervenes.

We cannot escape the organism except in avoiding contact with sufferers, but much can be done to avoid colds, even in

these cases, by attention to factors contributing to colds (exposure, lowered resistance, etc.).

If, from the history or other data, we find our patient suffering from protein sensitization, we are able to test him out and in many instances definitely prove the causative factor. When that is done, much can be promised in the way of relief by proper immunization.

Focal infections have been the cause of so many obscure conditions that it is not surprising to find many nasal conditions clear up upon the removal of that cause. A patient may have more than one focus, and, when all are removed, we have all seen improvement, just as Sawtelle describes. Vaccine therapy will be beneficial in certain cases.

Colds coming with acute disease clear up with the disease. Those resulting from the more chronic systemic disease should be recognized and properly diagnosed to be relieved, especially those due to syphilis, tuberculosis, dyscrasia of the glands of internal secretion, cardiorenal disease, hepatic sclerosis, nervous diseases, etc., improvement depending on the treatment of the chronic disease more than on the local treatment.

The colds due to occupations are often relieved or stopped when the patient realizes the cause, and especially when the employer and the employee work together with the physician to lessen or relieve the situation.

The condition of the nose itself is of importance in colds. A patient with a badly obstructed nose may have very few or almost no colds. He may have high natural immunity, good general resistance, and be vigorous enough to escape, but in one susceptible to colds an obstructed nose or one with unequal passageways there is better ground for a cold than where the nose is normal.

Further, there is the possibility of some infection locally. Some previous cold in the head has left one or more of the sinuses chronically involved. The particular sinus may not be obstructed in many of these cases, but the infective organism is present in the nose. Any indiscretion (chilling or accidental lowering of resistance) brings on the acute head cold. Instead of establishing immunity, the susceptibility increases. The majority of chronic sufferers from sinus infection have nasal obstruction. Some one or more of the sinuses are partially

blocked. Engorgement of the membrane blocks the sinus and keeps the air out, as well as allowing the secretion incident to the swelling of the mucous membrane no escape. This stagnation results in infection and injury to the sinus mucous membrane.

The trouble is, and has been so many times, that we get an anatomically normal nose, even free from infection, and, having removed every discernible cause of colds, yet patients return with as many colds as before. Between five and six years ago I was forcibly struck by the statements of two patients to the effect that neither of them had had a cold in ten years. I complimented them on being so lucky. Neither of them considered it luck. One said it was accomplished by a cold tub bath every morning. Instead of being disagreeable, it was a pleasure to jump in, and susceptibility to colds had disappeared. The other patient accomplished the same result by standing in front of an open window, looking out against a blank wall, where the wind would blow through in winter and summer. The morning air bath had practically eliminated colds. This impressed me that something might be done in these cases, and I began questioning my patients to see if I could find out what in their experience had brought on their colds. In a large number I found what I termed a skin complication, no matter whether there was an underlying condition, such as tonsils, sinuses, a dietary indiscretion, or even in epidemics. There was usually some chilling associated with it. They were in a draft, had hurried and become too hot, or got their feet wet and then "caught cold."

I always try to eliminate all etiologic causes and relieve the nose from obstruction, and then instruct those who are still susceptible to colds to stimulate the skin reaction by using cold baths or showers or air baths. I believe the trouble is that the skin does not react quickly enough in those susceptible. Obviously, the skin, through the heat center, is the real protective organ. Our mode of living has left less and less to the skin to perform, and its activity needs stimulation and use just as our muscles and teeth do. We have warm rooms to live in, many get up in a warm room, and we change our clothing at each change in temperature until the skin acts sluggishly in preventing loss of heat or in keeping us warm. The object

of stimulation is not to get chilled but to get skin reaction. It would be wrong to jump into a cold tub until one is trained to it, just as it would be wrong to exercise to exhaustion the first day in calisthenics. One should at first have cold water run only on the wrists, the next day on the arms, then on the limbs, each day going a little farther until one can use the cold spray over the whole body. It is as difficult to get one's patients to do this every morning as it is to get them to take exercise every day, unless they have a regular class of instruction. I believe the best results are obtained without the hot bath first. My limited experience thoroughly convinces me that it is a very practical and useful help. May I report a few typical cases?

One patient was very susceptible to acute colds, with laryngitis. He could be certain of having one each week, sometimes two. Coughing would keep him awake at night, two or three nights a week. An ice cap on his throat gave him the most relief, and he kept one in his grip for fear he would leave home without it. He could always count on the slightest draft, running for a car, getting moist or slightly fatigued to cause an attack. One antrum was found to be infected. Drainage gave some improvement but very little. The other antrum became infected during one of the attacks and that had to be drained. The colds were just about as numerous. In desperation the cold sprays were started, first on the wrists, then a little farther each day until the cold water could be turned on the whole body. The number of colds began to lessen so that in the past two years he has had about four colds, and it is almost a year since he has used the ice cap on his throat.

Two patients, one a girl, the other a boy, were continually catching cold. Both had bad tonsils and adenoids, which were removed. In one it was believed that the adenoids had returned, and a second operation was performed, almost nothing being found. The symptoms of catching cold continued. The cold showers were tried and improvement has been satisfactory. It was necessary to persist for some months with the boy before results were obtained.

That, I believe, is the reason why so little has been accomplished by these measures. The physician does not get quick

enough results, and the patient does not wish to do anything disagreeable, so it is not given a fair trial.

Another patient, an elderly lady, suffered from bronchitis after coryzas. The bathing of her chest and throat each morning in cold water increased her resistance so that the first winter she had less than one-half her usual number of colds and all seemed of shorter duration.

Needless to say, many patients will not consent to try. Many have taken only an occasional cold shower.

I began these observations several years before seeing the articles quoted in this paper. It seems to me that Mudd, Goldman and Grant have proved more than they claim. Their experiments were with young, vigorous subjects whose heat regulation mechanism was, so to speak, superactive, and yet they did produce colds and sore throat among a number of the subjects. What would have been the clinical result had they subjected some of the susceptible patients we all see to the chilling process? I am quite certain their clinical results would have been some very sick patients. Their subjects had the resistance, the ideal skin response which we should try to develop in our patients.

Mudd, Goldman and Grant believe in the hardening possibility in prophylaxis, but advise a warm bath before a cold one. Stoker advises fresh air, a daily bath, exercise, attention to excretory functions and foods rich in vitamins. Ferreri advises cold morning baths and habitual exposure of the throat. Max Gahwyler advises gradual hardening, with special care in children of lymphatic, arthritic or neuropathic families. Berry advises that various hardening processes should be used but that the correction of faulty hygiene and avoidance of contact infection should be kept in mind. Jordan, Norton and Sharp, in their investigations among students, thought resistance building practice had little effect on the frequency of colds. Theirs is the only article which is skeptical about that point, and the question naturally arises as to how thoroughly they were practiced, because one cannot be prepared for any test of endurance or resistance unless one keeps in training. It is of interest to note that 64 per cent believed their colds were induced by some strain on the heat regulating mechanism of the body.

Smiley, in his study of acute infections of the throat and respiratory system, pessimistically says, "Until the etiology of the infections is definitely settled, a specific prophylactic cannot be looked for." It seems to me that epitomizes the feeling of many men toward this subject. It is against that feeling I wish to protest. Just because we cannot at the moment place the etiology of all colds in a single category, with a precise prophylaxis and a more precise treatment, it is no reason for telling ourselves and our patients that we have nothing to offer in relief. Each case must be studied by itself to determine its needs.

The environment, the places we frequent, our homes, theaters, churches and schools are of the utmost importance. Leonard Hill, in his monograph, emphasizes the deleterious effect of going from an illy ventilated room into the cold outdoor atmosphere. Palmer, in "Ventilation, Weather and the Common Cold," concludes, in studying ventilation in schools, that window ventilation, with natural stimulus of an atmosphere varying in temperature and motion, is superior to forced draft where the temperature is too high and uniform. In other words, it is best when the skin is stimulated by proper movement of air and change in temperature.

One way to keep the skin properly active is to allow a certain portion of it free access to the air. One should instruct sufferers to habitually expose the throat to the air, as Ferreri suggests. It should never be covered, unless in long severe exposure. The bare knees in children are advantageous, only too many times they are covered one day and not covered the next. The rest of the body should be warmly clad but never so that perspiration begins. In many cases one finds susceptibility to colds due to houses being too warm, and especially too dry, with lack of proper air circulation.

Prophylaxis, in short, is: Advise the avoidance of contact with sufferers. Study each case to find and eliminate any and all factors in the etiology of colds in that case. Study the environment of the patient to improve, whenever possible, in his home, his work and his recreation. Advise him as to health habits in diet, elimination, work, play, etc., and especially get him to train the skin reaction.

Every writer has his own particular preference in treatment. It is to be noted, however, that silver in some form is almost universally used for local application or instillation.

Stoker gives 15 to 20 gr. calcium lactate t. i. d., and small amounts of atropin sulphate. He mentions some are for and some against cauterizing the septum tubercle with phenol and painting the vestibule with strong silver nitrate.

Ferreri recommends salicylate of quinin; Dover's powders, or pyramidon; potassium bromid and belladonna in children. Locally, zinc and aluminum bismuth sprays; cocain, adrenalin in oily base.

Chatin warns that menthol may cause fatal spasm in infants. He uses 10 per cent argyrol dropped into the eyes three or more times a day in children.

Botner is very enthusiastic about using 2 per cent collargol, dropping it into the conjunctival sac and nares, believing that swallowing some is very beneficial.

Isaacson obtained good results with lenigallol locally.

M. Groseffi uses 10 per cent protargol on nasal mucosa.

Max Gahwyler uses iodids to promote perspiration and elimination.

Boebinger opens the nose with cocain and adrenalin and uses silver nitrate or argyrol locally and inhalations of menthol and tincture of benzoin in hot water. He uses hot or cold compresses to forehead and eyes.

Benson Wood uses the quartz light locally on the mucous membrane with good results.

Vedder and Sawyer use chlorin as a therapeutic agent.

Any of these remedies will be found to be useful, and in practice the treatment of these conditions depends on when one is able to prescribe.

Usually is it too late to abort a cold. When seen immediately after the chilling, when that is a factor, a large number of colds can be aborted by immediately getting the patient to bed and perspiring freely. (Hot water bottles, hot drinks and blankets). This probably brings the temperature of the blood slightly above normal and allows a readjustment of the heat mechanism, with beneficial changes in circulation and skin elimination. When seen later, a brisk cathartic, hot bath on going to bed, salicylates, alkalization with sodium bicarbonate

and an atropin combination to lessen the nasal secretion and open the nose, silver nitrate locally to use once a day, with a nasal ointment or spray to use at home, with instructions to blow the nose gently, one nostril at a time, are of importance.

As a rule, a little care early during the acute attack quickly establishes drainage and recovery. When not relieved, swelling becomes chronic. Usually a sinus is involved, and more energetic measures are necessary to get relief. After the acute attack, we must recall the best treatment is prophylaxis. For emphasis I repeat that Mudd, Goldman and Grant, Stoker, Ferreri, Gahwyler and Berry suggest the possibility of benefit in a hardening process; that Jordan, in one of his studies, found 64 per cent thought their colds due to strain on the heat regulating mechanism of the body; that from the cases I have observed I am certain the skin can be trained to react more promptly and furnish resistance more efficaciously against cold than other factors under our control.

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507 PHOENIX BLDG.

XCVIII.

EXTRANASAL VS. INTRANASAL OPERATION FOR
CHRONIC SUPPURATIVE FRONTAL
SINUSITIS.*

By FRED STAUFFER, M. D.,

SALT LAKE CITY.

This paper deals with the subject of operation for chronic suppurative frontal sinusitis, and therefore I wish to exclude from the discussion any operation or treatment for acute or subacute suppurations in these sinuses. I have for some time had a very decided opinion as to the proper procedure in these cases; but, for the purpose of getting the viewpoint of others, I sent out a questionnaire to two hundred members of the Pacific Coast Oto-Ophthalmological Society who are practicing in communities with similar conditions as prevail in our own locality.

I received ninety replies, twenty-five of these from men who limit their practice to diseases of the eye, therefore only sixty-five doctors doing sinus work replied to the following questions, viz.:

1. Is your preference for intranasal or extranasal operation?
2. What extranasal operation do you prefer?
3. Does size or shape of sinus, as revealed in skiagram, influence you in your choice of operation?

To question No. 1, about forty expressed preference for intranasal, eighteen for extranasal and the balance did not believe in any operation. I observed that those who expressed a preference for the extranasal operation were very pronounced in their choice, while most of those who gave first choice to intranasal operation qualified their remarks by the statement that if the intranasal operation failed they would resort to the extranasal method.

In answer to the second question, thirty preferred the Kilian operation or some of its modifications; eleven were out-

*Read before the Mid-West Section of American Laryngological, Rhinological and Otological Society at Colorado Springs, Feb. 14, 1925.

spoken for the Lathrop method, and four preferred the Knapp operation, which is also claimed by Lynch and Barany. The balance had no choice.

In answer to the third question, nineteen said yes, and the rest paid no attention to size or shape of sinus in deciding upon the method of treatment.

From analyzing the comments that were made on question No. 1, I am sure that many of the doctors who expressed preference for the intranasal operation had in mind more or less acute, or at least subacute suppurations, and not strictly chronic cases. At any rate, I am of the opinion that the majority of the men who replied to my questionnaire would do just as I do in the same kind of cases, viz., the extranasal operation. We have for many years made skiagrams of all our chronic sinus suppurations and have thereby verified the facts so well brought out by Cunningham, Shaefer and others, viz., that the frontal sinuses differ greatly in size and contour, not only in different individuals, but that they are rarely the same size on both sides in any subject. We shall show a few slides which will emphasize the points we wish to bring out in our contention for the extranasal operation as a method of choice in all chronic suppurations. Two years ago I had the pleasure of taking a course under Dr. Barany at Los Angeles, where I saw him demonstrate an operation which he claims to have developed two years before Dr. Lynch of New Orleans wrote up the same technic, having independently developed the same principle, while someone unknown to him had done practically the same operation two years before Barany. I believe with Barany that the internal operation is dangerous and inefficient in the majority of chronic cases.

I am therefore strong for the external operation. In this, my choice is largely influenced by the size and contour of the sinuses as shown by the skiagram. There are very few cases, I believe, which require the disfiguring operation of Killian. They are those with very large extensions upwards and laterally. Such sinuses are difficult to cure permanently of discharge without complete obliteration, as is most satisfactorily effected by the Killian operation; and in these shallow cases there is very little deformity by the Killian method. Where

the sinuses are deep anterioposteriorly almost any extranasal operation which permits the cleaning out of mucous polypi and breaking down septi and enlarging the frontonasal passage will give satisfactory results. In cases where both sinuses are involved the Lathrop operation should be the method of choice. In anterioposterior deep cases, with only one sinus involved, the Ogsten-Luc operation offers a simple and effective cure. In a large number of cases, where there are many septi and cells extending well back over the roof of the orbit, sometimes as far back as its apex, the Knapp-Lynch-Barany operation is the method of choice. Barany describes the operation as follows:

He prefers local anesthetic of 1 per cent novocain, with 15 drops of 1/1,000 adrenalin to the ounce. This he injects freely into the skin and cellular tissue over the sinus and side of the nose, until the skin becomes tense over the eyelid and forehead. Also blocks nasal ganglion after Sluder method. He does not inject any anesthetic into the orbit. Makes incision through the unshaven brow and down the inner margin of the orbit just as in Killian operation. The skin and periosteum are carefully dissected downward, taking care to separate the periosteum from the supraorbital ridge and roof of orbit without puncturing it, if possible. Also separates the periosteum and lacrimal sac from the inner wall of the orbit. Makes opening into sinus at inner lower angle; removes whole floor of sinus, part of the nasal and lacrimal bones, and inner wall of orbit back to apex if necessary; cures out all diseased mucosa in the sinus, removes part of middle turbinal and all anterior ethmoid cells, but leaves the inner plate of ethmoid labyrinth so as to avoid injury to cribriform plate. The skin wound is completely closed; a short probe wrapped with sterafil paper or rubber tissue is passed into sinus through the nose and held in place by adhesive plaster. This is changed and nose cleansed daily for five or six weeks, when the case is usually healed. This operation causes no disfigurement, very little pain in after treatment, and because of its complete obliteration of all cells and septi is most effective so far as cure is concerned.

Two years ago, Dr. Lynch reported forty-two operations with only two failures. This method has been very successful

in our cases. My own experience with external operations, over a period of twenty-five years, has been limited to the Ogsten-Luc, the Killian and the Lynch-Barany methods. In our early cases, before the general use of the skiagram to determine the size and shape of these sinuses, we used to do many Ogsten-Luc operations with more than 75 per cent cured. Later, because of failure with this simple operation, we resorted to the Killian method, not regarding the great deformity which results in those deep anterioposterior sinuses, because of the uniformly good results which were obtained. During the last two years we have followed only the Barany method with great satisfaction. The slides which follow, I think, will clearly justify the contention that any internal operation is ineffective and dangerous in a great many of these cases. The more I practice in surgery, the more I am impressed with the importance of exposing to view the field of operation, and not groping in the dark, especially when working in a septic field so near the brain and its coverings.

By way of summary, let us observe the points I wish to emphasize:

First—The open or extranasal operation is the method of choice in chronic suppurative frontal sinusitis, because one has clear view of the pathology, and therefore better able to thoroughly eradicate the diseased parts.

Second—The choice of open operations is influenced by the size and shape of sinus, as shown by skiagram. The extensive but shallow sinuses are most effectively treated by the Killian method, while the anteroposterior deep sinuses without septi respond favorably to the simple Ogsten-Luc operation, and the Barany method is successful and least disfiguring in all other sinuses.

801 DESERET BANK BUILDING.

XCIX.

THE OPTIC CANAL IN OPTIC ATROPHY.

BY LEON E. WHITE, M. D.,

BOSTON.

As this paper is the third devoted to the investigation of optic canals, a brief resume of the preceding ones seems in order.

ANATOMIC.

In an examination of about two hundred skulls the diameter of the optic canals was found to vary from 4 to 6 mm., the average being 5.17 mm., and the shape from the usually circular to various degrees of ovalness. Both size and shape depend upon the pneumatization within the accessory sinuses adjacent to the canals, the greater the pneumatization the smaller and more irregular they become. It seems probable that the neural contents of these small excessively pneumatized canals would be especially vulnerable, not only because they are more completely surrounded by the mucosa of the adjacent sinuses but also because their bony walls are thinner and offer a lesser barrier to infections. Clinical data has fairly well borne out this belief.

RADIOGRAPHS.

The true size and shape of the optic canal can only be obtained when its axis is close to the lower outer quadrant of the orbit. The optic canal in the living can be thrown into this area when the patient is lying face down with the malar bone, nose and lower jaw touching the plate, the central ray directed straight down.

The diameter of the optic canal, as it appears on the radiographic plate, is enlarged about 10 per cent, but as this is the only method of determining its size no deductions are made in recording cases. The relative sizes are all that are needed. The normal canal in the radiograph is practically 5.5 mm. The average diameter, as obtained by filming twenty-five supposedly normal individuals, was 5.35 mm., and of these 10 per

cent were oval. These canals were contrasted with an equal number from patients with optic nerve involvement, where the average diameter was found to be $\frac{2}{3}$ mm. less, and in 50 per cent of these the canals were oval.

The conclusions from this anatomic and radiographic study are that if future cases substantiate the results of those analyzed, one can have a valuable guide as to the necessity for surgical intervention in cases of retrobulbar and optic neuritis.

TREATMENT.

In a second paper an attempt was made to ascertain what influence the size and shape of the canal might have on treatment. This was done by analyzing thirty-six cases previously treated for some form of optic nerve involvement. It was found that in the small canals—i. e., 4 mm. or less—there was 50 per cent in which the loss of vision was permanent, while in the large canals this percentage was considerably smaller.

During the past two years an effort has been made to either substantiate or disprove these views. It seemed to me that by measuring the canals in a large series of optic atrophy cases some valuable data could be acquired. Accordingly, through the hearty cooperation of the social service, the ophthalmic staff, the X-ray department of the Massachusetts Eye and Ear Infirmary and the State Department of the Blind, many patients with optic atrophy were investigated. I personally made the nose and throat examination. The fundi were examined by a member of the ophthalmic staff, usually Doctor Joseph Ellis, and the optic canals, sella turcica and accessory sinuses were filmed by Doctor A. S. Macmillan. Careful records were made by the social service, and at the completion of the work I analyzed the records made by the various examiners and have incorporated them in this paper. Many of the cases, while diagnosed as optic atrophy, had other eye conditions, but nearly all were helpful for comparison. While the main object of this investigation was to establish the relationship, if any, between the size of the canal and the loss of vision, another aim was to determine whether the canals became enlarged from increased intracranial pressure or narrowed when the nerve atrophied.

Sixty-seven cases of optic atrophy, mostly from the Perkins Institution for the Blind, were studied; of these, ten were

unilateral. This gives 124 optic canals, which are subdivided into five groups according to area. Occasionally the canals in a case differ so much in size that they belong in different groups. Either from the history of these cases or from previous records it has usually been possible to determine the cause of the atrophy. Whenever we have a specific history or find Hutchinsonian teeth the atrophy is classified as of luetic origin, in spite of the fact that in many of these, infected sinuses, tonsils or teeth might have been a contributing factor. Many have had brain tumors removed or the radiographs show an enlarged sella. Here the atrophy is, in all probability, due to the intracranial condition, and the nose and throat pathology is but an incident.

Cases which have been blind since birth or early infancy, where the causes are not generally known, are classified as congenital. In these the nose and throat could not have been in any way a causative factor. In other cases the atrophy is the terminal result of a retinitis pigmentosa, and the nasal condition has no bearing on the etiology. In some of the cases, however, the history is so typical of a retrobulbar or optic neuritis, either from nasal, dental or tonsillar infection, that there is little hesitation in classifying the atrophy as secondary to the neuritis. Several of these cases have, as a matter of fact, been followed from the acute onset to complete atrophy. Ninety-three, or 75 per cent, of the 124 canals associated with optic atrophy were below normal. Twenty-two, or 18 per cent, were normal. Eleven, or 7 per cent, were above normal.

GROUP I.

This contains the very small canals rarely, if ever, seen with normal vision. Their area is from $\frac{1}{3}$ to $\frac{1}{2}$ the normal. In this group is classed the 3, 3.5, 3.5 x 4, 3.5 x 5 and 4 mm. canals, thirty in all (see Group I).

Summary of these 30 canals:

In 9 the atrophy was secondary to a retrobulbar neuritis of nasal origin. All had obstruction to the posterior sinuses.

In 5 the atrophy was secondary to lues. All had nasal blocking and suspicious tonsils.

In 6 the atrophy was secondary to brain tumors. Four had nasal blocking or infected sinuses. Two had infected tonsils.

In 6 the atrophy was congenital. Nose negative in all. Hypertrophied tonsils in two.

In 2 the atrophy was secondary to retinitis pigmentosa. Nose and throat normal.

In 2 the atrophy was secondary to spinal meningitis. Hypertrophied tonsils.

Comment.—In eighteen of these thirty canals there was marked nasal pathology, which in nine was considered to be the cause of the atrophy. While hypertrophied tonsils were found in eleven, in none were they considered to be a factor in the loss of vision.

GROUP II.

These canals are somewhat larger than those in the preceding group, and are found in cases with normal eyesight. Their small size, however, is a distinct menace to vision, when their neural contents are involved in any inflammatory process. They are from $\frac{2}{3}$ to $\frac{3}{4}$ of the normal size and comprise the 4×4.5 , 4×5 , 4.5 , 4.5×5 mm. canals, forty-one in all (See Group II).

Summary of these 41 canals:

In 3 the atrophy was secondary to retrobulbar neuritis from nasal infection. Infected sinuses.

In 6 the atrophy was secondary to chronic retrobulbar neuritis from dental infection. Nose negative in all. Suspicious tonsils in three.

In nine the atrophy was secondary to lues. Nasal obstruction in 7. Tonsils suspicious in 5.

In 4 the atrophy was congenital. In 2 pansinusitis, nasal obstruction and diseased tonsils.

In 4 the atrophy was secondary to retinitis pigmentosa. In 2 nasal blocking and infected tonsil stumps.

In 2 the atrophy was secondary to infantile paralysis. Nasal obstruction, pansinusitis, infected teeth and tonsils.

In 4 the atrophy was secondary to fractured skulls. Nasal obstruction in all.

In 7 the atrophy was secondary to brain tumors. Nasal obstruction in 5. Diseased tonsils in 3.

In 2 the atrophy was secondary to meningitis. Nose and throat negative.

Comment.—In but three of these canals was nasal infection the probable cause of the atrophy, while infected teeth appeared to be responsible for the atrophy in six canals. The nasal obstruction and tonsillar infection found in most of the other cases were probably not responsible for the loss of vision.

GROUP III.

These canals are from $3/4$ to $9/10$ of the normal size and include the 4.5×5.5 , 4×6.5 , 5 and 5×5.5 mm. canals, twenty-two in all (See Group III).

Summary of these 22 canals:

In 2 the atrophy was secondary to multiple sclerosis. Suspicious tonsils.

In 4 the atrophy was secondary to chronic retrobulbar neuritis of dental origin. Diseased tonsils.

In 2 the atrophy was secondary to chronic retrobulbar neuritis from diseased tonsils. Infected tonsils.

In 6 the atrophy was congenital. Nasal obstruction and diseased tonsils in 2.

In 2 the atrophy was secondary to brain tumor. Nasal obstruction, enlarged tonsils.

In 2 the atrophy was secondary to infantile paralysis. Nose and throat negative.

In 2 the atrophy was unclassified. Nose and throat negative.

In 2 unilateral cases the atrophy was secondary to acute retrobulbar neuritis, probably from infected tonsils. Nasal obstruction.

Comment.—In this group there are but two canals (unilateral cases) where the atrophy was thought to be secondary to an acute retrobulbar neuritis, probably of tonsillar origin, although in both there was some nasal obstruction.

There were six canals (3 bilateral cases) where the atrophy appeared to be secondary to chronic retrobulbar neuritis of dental origin, although two of these also had suspicious tonsils.

In the other cases the nasal obstruction and suspicious tonsils did not seem to be a factor in the atrophy.

GROUP IV.

These canals, twenty in number, are of normal size, the measurements being 5.5 and 5×6 mm. (See Group IV).

Summary of these 20 canals:

In 4 canals the atrophy was secondary to lues. Nasal infection in 2. Dental infection in 2.

In 2 the atrophy was secondary to pituitary disease. Nasal obstruction.

In 2 the atrophy was secondary to multiple sclerosis. Nose and throat negative.

In 2 the atrophy was secondary to cerebral hemorrhage. Infected teeth and tonsils.

In 2 the atrophy was secondary to retinitis pigmentosa. Slight nasal obstruction.

In 2 the atrophy was secondary to oxycephaly. Nasal obstruction.

In 2 the atrophy was congenital. Nasal obstruction.

In 4 (unilateral) the atrophy was secondary to retrobulbar neuritis, probably of dental origin. Nasal obstruction in 2, suspicious tonsils in 3.

Comment.—There were but four cases in this series where the atrophy seemed to be secondary to retrobulbar neuritis, and in three of these at least it appeared to be of dental origin. In most of the other cases the pathology found in the nose and throat did not appear to be a factor in the loss of vision.

GROUP V.

These canals are above normal, eleven in all, the measurements being 5 x 7, 5.5 x 6, 5.5 x 6.5, 6, 6 x 7 and 7 mm. (See Group V).

Summary of these 11 canals:

In 6 the atrophy was secondary to brain tumor. Nose and throat negative in 4. Diseased tonsils and nasal obstruction in 2.

In 4 the atrophy was secondary to oxycephaly. Nose and throat negative in all.

In 1 the atrophy was secondary to pituitary disease. Nasal obstruction and suspicious tonsils.

Comment.—Associated with three of these eleven canals there were diseased tonsils and nasal obstruction. Atrophy in all, however, was due to the intracranial disease.

LARGE OPTIC CANALS.

In all the canals above normal there was intracranial involvement—i. e., there were 6 canals in brain tumor cases, 1 in a pituitary and 4 in tower skulls. The evidence from these few

cases, therefore, indicates that increased intracranial pressure may produce a dilatation of the optic canals. The cause of this dilatation is probably the continuous pressure of the spinal fluid within the optic nerve sheath against the walls of the canal.

Intracranial lesions were responsible for the atrophy in but 16 per cent of the canals below normal.

When we contrast this 16 per cent with 100 per cent in the abnormally large canals we have facts difficult of interpretation unless we concede that increased intracranial pressure produces dilatation of the canals.

Of the twenty canals of normal size the atrophy in two was due to pituitary disease and in two to oxycephaly. In neither of these cases did the intracranial pressure seem to be as marked as in the cases with abnormally large canals, this opinion being based on the smaller size of the sella turcica, as compared with the sellas in the larger canal cases.

As previously stated, 16 per cent of the cases with canals below normal had intracranial lesions. This shows that brain tumors do not necessarily produce dilatation of the canals. It is probable that the papilledema was either of short duration or absent, for, according to Gowers and Barker, papilledema is not present in from 10 to 20 per cent of brain tumor cases.

SMALL OPTIC CANALS.

The average diameter of twenty-eight canals in cases with congenital atrophy was 4.5 mm., which is approximately the average in the entire series, so that as far as this data goes early atrophy of the nerve does not account for the small canals. While small canals seem to be a distinct factor in the production of atrophy, early atrophy does not explain their diminished size.

The practical point in this study is the question of enlarging the optic canal should one of these small canal cases be seen before the atrophy is complete. The canal can be approached by three routes—i. e., the frontal way, as advised by Schloffer; the orbital route, as performed by Hildebrand, and the lateral route, the method employed by Dandy. Van der Hoeve, in a recent article on optic nerve tumors, says: "Operations by the lateral way give a liberal view of the condition at the intracranial optic foramen and allow us to enlarge the optic canal.

. . . . This operation can be used if the canal is considerably deformed or absolutely too narrow—i. e., in cases of optic nerve involvement, as tower skull fractures, etc."

He also adds that "the canal operation for preservation of the visual power should be performed only if both optic nerves are endangered in such a measure that absolute blindness is to be feared."

These operations on the optic canal are not devoid of danger; they have been but rarely performed and would require all the skill and dexterity that come through long training in neural surgery. While I have never seen a case in the stage when it could have been beneficial, there are probably several in this series where it might have prevented the atrophy.

CONCLUSIONS.

In many of these cases, where the atrophy was secondary to various other conditions, there was nasal obstruction, infected sinuses, teeth or tonsils. This fact needs emphasis, as it is only too common a failing among some rhinologists to attribute to pathology in their own province most of the optic nerve disturbances.

Whatever scientific value this paper may have will be found in the comparison between the sizes of the optic canals in these various groups, and the sizes from a large number of normal cases similarly grouped. Fortunately, this comparison could be made, as I had the data from the measurements of 300 canals in skulls at the Harvard Medical School.

Twenty-one per cent of all the optic atrophy canals was in Group I, these being the very small canals rarely, if ever, found with normal vision, while in the medical school series, which should represent the normal, there was 1 per cent in Group I.

There can be little doubt that a proportion so marked as 24 to 1 can be interpreted in any other way than that the predisposition to permanent loss of vision is markedly increased when any process capable of producing atrophy occurs within these abnormally small canals.

Thirty-three per cent of the canals was in Group II, as contrasted with 7 per cent found in the medical school measure-

ments. Here again, with a proportion of 7 to 33, the diminished area of these canals contributes without doubt to the high percentage of atrophy, although not to the same extent as in Group I.

Thirty-four per cent of the optic canals was in Groups III and IV, these being the normal canals and those but slightly below normal. According to the medical school series there should have been 85 per cent of all the canals in these groups. This clearly indicates that the canals, as they approach the normal size, rapidly cease to be a menace to their neural contents. Immunity to atrophy, however, is not found in any sized canals.

In the canals above normal (Group V) it was interesting to find how closely the two series compared. Thus, there should have been 7 per cent, according to the medical school series, while as a matter of fact there was 8 per cent in the optic atrophy group. This seems to indicate that in a certain small percentage of cases the size of the canal is not a factor in the atrophy. In these particular cases it was due to increased intracranial pressure.

SUMMARY.

This is the third paper devoted to the study of optic canals. In the first it was shown that excessive pneumatization of the sinuses adjacent to the canals produced narrowing and distortion. In a series of radiographs it was found that patients with optic nerve disturbances had abnormally small canals. By analyzing thirty-six cases of optic nerve involvement it was found that in the smaller canals the vision in 50 per cent was permanently impaired, while in patients with larger canals the percentage was much less.

Sixty-seven cases of optic atrophy were studied. These were divided into five groups, according to the size of the optic canal. Nasal infection was responsible for atrophy in twelve out of one hundred and twenty-four canals, or 10 per cent. This occurred in the small canals—nine in Group I and three in Group II. The other causes were as follows:

In 24 canals the atrophy followed brain tumors or pituitary disease.

In 18 canals the atrophy was of luetic origin.

- In 18 canals the atrophy was congenital.
- In 14 canals the atrophy was from diseased teeth.
- In 8 canals the atrophy followed retinitis pigmentosa.
- In 6 canals the atrophy was from oxycephaly.
- In 4 canals the atrophy was secondary to tonsillar infection.
- In 4 canals the atrophy was from spinal meningitis.
- In 4 canals the atrophy was from infantile paralysis.
- In 4 canals the atrophy was from multiple sclerosis.
- In 2 canals the atrophy followed cerebral hemorrhage.
- In 2 canals the atrophy was unclassified.

Increased intracranial pressure seemed to be responsible for the abnormally large optic canals, as intracranial lesions were found in all. Atrophy of the optic nerve in infancy was not responsible for the very small canals.

While it is possible to enlarge the optic canal surgically, cases are rarely seen at the stage when it would be beneficial.

In comparing sizes of optic canals in these atrophy cases with the normals, it was found that in Group I the proportion was 24 to 1, so there would seem little doubt that a proportion so marked as this could be interpreted in any other way than that the predisposition to permanent loss of vision is markedly increased whenever any process capable of producing atrophy occurs in abnormally small canals. As the canals approach normal size they rapidly cease to menace their neural contents, although atrophy may occur in canals of all sizes.

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390 COMMONWEALTH AVENUE.

NAME AND AGE AND X-RAY No.			HISTORY		EYE CONDITION		NOSE, THROAT AND TEETH		RADIOGRAPHS OF SINUSES AND OPTIC CANALS	
CE	X-RAY No.	AGE	HISTORY		EYE CONDITION		NOSE, THROAT AND TEETH		RADIOGRAPHS OF SINUSES AND OPTIC CANALS	
	6507	7	Congenital. Never saw anything but light perception.		on optic atrophy. Ptm. m. V. on light perception.		Large T & A*. No blocking.		Sella normal. Sinuses negative. Both 4 mm. irregular.	
DT	6608	13	P I	Born blind. Congenital lues?	on optic atrophy. V nil.		Diseased tonsils. Deflected septum. Purulent rhinitis. Nasal blocking.		Sella normal. Sinuses negative. Excessive pneumatization left wing sphenoid. Rt. 4 mm. Sella normal. Sinuses negative. Sphenoid very large. Excessive pneumatization extending into lesser wing left.	
MJ	6241	17	P I	Congenital lues. Hutchinsonian teeth.	on optic atrophy. V nil.		Large tonsils. Deflected septum. Nasal blocking.		Sella normal. Sinuses negative. Rt. 3.5 flattened on top. Rt. 4 mm.	
PM	7643	12	P I	Sudden loss of vision over night 7 years ago.	on optic atrophy. V nil.		Deflected septum. Some blocking.		Sella normal. Sinuses negative. Both 3.5 mm. irregular. Increased density at periphery.	
TH	8642	14	P I	Brain tumor? Lost vision 9 years ago.	on optic atrophy secondary. V od F* 5 ft. on nil.		Large T & A. Deflected septum. Hypertrophied mid. turbinate. Blocking.		Sella normal. Left antrum cloudy. Sella very small. Sinuses negative. Both 3.5 mm.	
MCH	6239	11	P I	Vision lost 4 years ago. Hutchinsonian teeth. Congenital SP?	on optic atrophy. V light projection.		T & A enlarged. Deflected septum. Enlarged mid. turbs. Blocking.		Sella normal. Sinuses negative. Rt. (See Group 2). Lt. 4 mm.	
SM	7359	7	P I	Gradual loss of vision for 3 years.	on optic atrophy primary? V light projection.		Enlarged mid. turbinate. Deflected septum. Slight blocking.		Sella normal. Sinuses negative. Both 3.5 x 4 mm.	
CE	27	16	Priv.	Optic neuritis followed by complete atrophy, first in one eye and 6 months later in other.	on optic atrophy. V nil.		Marked hypertrophy both mid. turbs. Marked obstruction both sides.		Sella normal. Sinuses negative. Both 3.5 x 4 mm.	
SH	7916	12	P I	Congenital blindness.	on microphthalmos. V nil.		Slight hypertrophy both mid. turbs. No blocking.		Sella normal. Sinuses negative. Both 3.5 x 4 mm. Flattened on top. Increased density.	
CB	7917	12	P I	Sudden loss vision 4 years ago. Cerebellar tumor removed.	on optic atrophy secondary. V nil.		Marked deflection septum. Hypertrophied turbs. Marked blocking.		Sella normal. Right antrum cloudy. Sella normal. Sinuses negative. Both 3 mm.	
RH	9170	10	P I	Blind since birth. Ophthalmia neonatorum.	on nasal side nerve pale, temporal side red. V od F 4 ft. on Lt. perception.		Several carious teeth.		Sella normal. Sinuses negative. Both 3 mm.	
NG	9167	21	P I	Practically blind since infancy.	on microphthalmos pigmentosa. V F few ft.		Negative.		Sella normal. Sinuses negative. Pneumatization of anterior clinoid. Lt. 3.5 x 5 mm.	
KC	8291	16	P I	Vision lost suddenly following spinal meningitis.	on optic atrophy secondary. V nil.		Enlarged tonsils. Deflected septum. No blocking.		Sella normal. Sinuses negative. Rt. 3.5 x 4.5 mm. Lt. 3.5 x 4 mm.	
SME	8226	17	P I	Vision lost when 7 years old. Brain tumor removed.	on optic atrophy secondary. V nil.		Deflected septum. No blocking.		Sella normal. Sinuses negative. Both 3 mm.	
DM	8624	21	P I	Vision lost suddenly following influenza 6 years ago.	on optic atrophy secondary. P 2 ft.		Deflected septum. Both mid. turbs. enlarged and blocking.		Sella normal. Sinuses negative. Rt. 4 mm. Lt. 3.5 x 4 mm.	
RME	8288	59	Inf*	Vision lost 7 years ago following scarlet fever.	on optic atrophy. V od F 15 inches, on shades.		Suspicious tonsils. Deflected septum. Some blocking.		Sella normal. Sinuses negative. Both 3 mm.	

GROUP II.

NAME AND XRAY No.	AGE AND INST.	HISTORY	EYE CONDITION	NOSE, THROAT AND TEETH	SINUSES AND OPTIC CANALS	RADIOGRAPHS OF
KT 8151	9 P 1	Poor vision since birth.	on optic atrophy. Chorioid changes V od F 6 inches, os F 3 ft.	Enlarged T & A. Deflected septum. No blocking.	Sella normal. Sinuses negative.	Both 4.5 mm. triangular.
AG 8290	18 P 1	Rapid loss vision. Cerebellar tumor removed.	on optic atrophy secondary. V od nil, os F 2 ft.	Enlarged tonsils. Deflected septum. No blocking.	Sella normal. Skull mottled. Sinuses negative.	Both 4 x 5 mm.
SH 8643	21 P 1	Gradual loss vision for 3 years. Wassermann positive.	on optic atrophy secondary. V od shadows.	Deflected septum. Some obstruction.	Sella normal. Sinuses negative.	Rt. 4 x 4.5 mm. Lft. 4.5 x 4 mm. v-shaped.
DA 8289	19 P 1	Sudden loss vision 15 years ago.	on optic atrophy primary. V od shadows, os F 6 inches.	Hypertrophied tonsils. Deflected septum. No blocking.	Skull mottled. Increased intracranial pressure. Ethmoiditis cloudy.	Rt. canal and surrounding structures destroyed. Lft. 5 x 4 mm.
JE 7641	21 P 1	Poor vision since infancy.	on optic atrophy. Congenital Sp. V od F 6 inches, os nil.	All negative.	Sella normal. Sinuses negative.	Both 4.5 x 5 mm. Increased density at periphery.
MacG R 8257	18 P 1	Blindness following meningitis 7 years ago.	on optic atrophy. V nil.	Slight hypertrophy left mid. turb. No blocking.	Sella normal. Sinuses negative.	Rt. 4.5 x 5 mm. Lft. 4 x 4.5 mm. Flattened on top.
CW 7918	11 P 1	Gradual loss vision. Cerebellar tumor removed.	on optic atrophy secondary. V light perception.	Deflected septum. Hypertrophied mid. turb. Slight blocking.	Sella normal. Sinuses negative.	Both 4.5 x 5 mm. Increased density at periphery.
JC 7769	40 Inf.	Sudden loss vision 2 years ago. Wassermann positive.	od optic atrophy. V od nil, os 20/20.	Diseased tonsils. Deflected septum. Hypertrophy right mid. turb.	Sella normal. Ethmoiditis cloudy.	Both 4.5 slightly irregular.
CA 7203	14 P 1	Sudden loss of vision 4 years ago.	on optic atrophy. Congenital lues? V objects, shadows.	Large T & A. Hypertrophied mid. turbulines with obstruction. Hutchinsonian teeth.	Sella normal. Sinuses negative.	Rt. 4.5 x 4 mm. Lft. 4.5 mm. Both irregular and increased density.
ARG 6741	8 P 1	Vision lost in infancy.	on optic atrophy. V objects, shadows.	Moderate T & A. Septum deflected. Mid. turbulines enlarged.	Sella normal. Pansinusitis.	Both 4 x 4.5 mm. Flat on top.
SG 6328	25 P 1	Vision poor since birth.	on optic atrophy. Retinitis pigmentosa. V objects.	Tonsil stumps. Deflected septum. Hypertrophied mid. turbulines with obstruction.	Sella normal. Sinuses negative. Pneumatization of bridge formed by lateral root of lesser wing.	Both 4.5 x 5 mm.
RTJ 11960	47 Inf.	Gradual loss left eye 2 yrs.	os optic atrophy. V od 20/40, os shadows.	Suspicious tonsils. Deflected septum. Hypertrophied left mid. turbulines. Interlobes with marked blocking.	Sella normal. Thickened membrane left atrium. Abnormal mid. Left sphenoid obscured.	Rt. 5.5 (normal). Lft. 4 x 5.
GE 4 6037	25 Inf.	Sudden loss of vision. Nearly blind in 6 weeks.	Acute retrobulbar neuritis. on optic atrophy. V F 2 ft.	Small tonsils. Slight enlargement mid. turb. Slight block ing.	Sella normal. Sinuses very pneumatic.	Both 4.5 x 5.
LL 6609	14 P 1	Gradual loss vision. Cerebellar tumor removed.	on optic atrophy. V od light perception, os nil.	T & A. Deflected septum. Moderate blocking.	Sella normal. Sinuses negative.	Both 4.5.
CP 6742	4 Inf.	Loss vision following fracture of skull.	F 12 ft. os F 4 ft. shadows, os 20/70.	Deflected septum. No blocking. Carious teeth.	Sella normal. Sinuses negative. Several abscessed ethmoids.	Rt. 4.5. Lft. 4 x 5. Somewhat irregular.
TH 7460	51 Inf.	Gradual loss vision for 20 years.	on optic atrophy. V od shadows, os 20/70.	Small tonsils. Deflected septum. Mucoid hypertrophy mid. turbulines. Blocking.	Sella normal. Thickened membrane frontal sinus.	Both 4.5 x 5. Oval with increased density.
DC 7453	52 Inf.	Gradual loss vision 10 yrs. Positive. Wassermann.	on optic atrophy. V od 12/200, os 8/200.			

* P 1 - Perkins Institution for the Blind. V - Vision. T & A - Tonsils and Adenoid. F - Fingers. Inf - Mass. Eye and Ear Infirmary.

GROUP II—Continued.

NAME AND X-RAY NO.	AGE AND INST.	HISTORY	EYE CONDITION	NOSE, THROAT AND TEETH	SINUSES AND OPTIC CANALS
SA 10284	42 Inf.	Gradual loss vision for 5 years.	on optic atrophy. V od F 2 ft. os F 10 ft.	Several abscessed teeth.	Sella normal. Sinuses negative. Abscessed teeth. Rt. 5 x 6 (Group IV), Lt. 4 x 4.5.
RC 9169	11 P I	Vision failed rapidly following infantile paralysis.	on optic atrophy. V ml. os light perception.	Enlarged T & A. Deflected septum. Carious teeth. Obstruction.	Sella normal. Translucid. Rt. 4 x 5. Lt. 4.5.
BA 9166	16 P I	Gradual loss vision after injury.	on optic atrophy. V ml. os shadows.	Deflected septum. Hypertrophied mid turbinate. Blocking.	Sella normal. Sinuses negative. Rt. 4.5 x 5. Lt. 4.5.
SR 9697	6 Inf.	Gradual loss vision for 1 yr.	on optic atrophy. V od 20/100. os 20/200.	Diseased tonsils. No blocking. Carious teeth.	Sella normal. Sinuses negative. Both 4 x 5.5.
SR 6238	27 P I	Poor vision since infancy.	on optic atrophy. Retin. pigmentosa. V	Small tonsils. No blocking.	Sella normal. Sinuses negative. Marked pneumatization about canal.
SM 7339	7 P I	Gradual loss vision 3 yrs.	on optic atrophy. V ml. os shadows. V light projection.	Slight deflection septum. Enlargement mid. turbinate.	Sella normal. Sinuses negative. Rt. 4 x 5. Lt. 4 (Group I).

GROUP III.

O'B T 12652	40 Inf.	Vision failed in left eye following accident.	on optic atrophy. V od 20/20. os 20/100.	Marked deflection septum. Hypertrophy left mid. turbinate. Blocking left.	Sella normal. Sinuses negative. Rt. 5.5 (normal). Lt. 5 x 5.5.
SM 6327	9 P I	Blind since infancy.	on optic atrophy. V ml. os shadows.	T & A. Deflected septum. Hypertrophied mid. turbinate. Marked blocking.	Sella normal. Sinuses negative; excessively pneumatized. Both very irregular.
DT 6608	13 P I	Blind since birth. Congenital lues.	on optic atrophy. V ml.	Diseased tonsils. Deflected septum. Purulent rhinitis.	Sella normal. Sinuses negative; excessively pneumatized. Both very irregular.
TB 24	40 Inf.	Sudden loss vision. Multiple sclerosis?	on optic atrophy. V 6 inches.	Nose negative. Suspicious tonsils.	Sella normal. Sinuses negative. Both 5.
FWR 46	35 Inf.	Gradual loss vision for 5 years. Slight improvement following removal infected teeth.	on optic atrophy. V 20/200.	Deflected septum. Hypertrophied mid. turbinate. Diseased tonsils.	Sella normal. Sinuses negative. Both 5.
McL B 48	39 Inf.	Gradual loss vision for 10 years. and complete loss vision. Rt. eye following acute retrobulbar neuritis.	on optic atrophy. V 10/200. os 20/200.	Suspicious tonsils. Abscessed teeth. Dental cyst. Deflected tonsil stump. Enlarged mid. turbinate with blocking.	Sella normal. Sinuses negative. Both 5.
AD 12597	18 Priv.	Gradual loss vision after infantile paralysis.	on optic atrophy. V ml. os 4/60.	Small tonsils. Both mid. turbinate hypertrophied. No blocking.	Sella normal. Sinuses negative. Rt. 5.5 x 4.5. Lt. 5.5 x 5. Flattened on top.
SD 5525	17 P I	Gradual loss vision for 3 or 4 years.	on optic atrophy. V od 20/70. os 20/200.	Infected tonsils. No blocking.	Sella normal. Sinuses negative. Both 5.
MH 10149	13 Inf.	Gradual loss vision for 2 years. Brain tumor removed.	on optic atrophy. V ml. os light perception.	Enlarged tonsils and mid. turbinate. Slight obstruction.	Sella normal. Sinuses negative. Both 5.
BM 10093	8 P I	Sudden loss vision.	on optic atrophy. V ml.	Deflected septum. No blocking.	Sella normal. Sinuses negative. Both 5.
LS 10052	8 P I	Poor vision since infancy.	on optic atrophy. V ml. os shadows. V F 4 ft.	Negative.	Sella normal. Sinuses negative. Both 5.5 x 5. Distorted, flattened laterally and on top.
MS 7360	7 P I	Blind since birth. Congenital lues.	on optic atrophy. V ml. os shadows. V F 4 ft.	T & A. Deflected septum. No blocking.	Sella normal. Sinuses negative. Both 5.
LMJ 7361	15 P I	Blind since birth. Congenital lues.	on optic atrophy. V ml. os shadows. V F 4 ft.	T & A. Deflected septum. No blocking.	Sella normal. Sinuses negative. Both 5.

GROUP IV.

NAME AND XRAY No.	AGE AND INST.	HISTORY	EYE CONDITION	NOSE, THROAT AND TEETH	SINUSES AND OPTIC CANALS
NE 7204	50 P I	Vision lost in 9 days when 17 years old.	on optic atrophy. V nil.	Dilated septum, hypertrophied mid. turbinates. Blocking.	Large sella (Pituitary abscess). Sinuses negative.
SA 10284	32 Inf.	Gradual loss vision for 5 years.	on optic atrophy. V od F 2 ft. os F 10 ft.	Abscessed teeth.	Sella normal. Sinuses negative. Abscessed teeth.
HA 8150	14 P I	Gradual loss vision when 2 years old.	on optic atrophy. V nil.	T & A. Deflected septum. Hypertrophied turbinates with obstruction. Hutchinsonian teeth.	Sella large. Skull shows evidence increased intracranial pressure. Oxycephaly. Sinuses negative.
BP 8152	17 P I	Gradual loss vision for 10 years.	on optic atrophy. Congenital lues. V 2/200.	Deflected septum. Hypertrophied mid. turbinate with obstruction.	Sella normal. Sinuses negative.
GR 7640	14 P I	Sudden loss vision 6 years ago. Positive Wassermann.	on optic atrophy. V od nil. os light perception.	Hypertrophied mid. turbinates. Punctate rhinitis. Carious teeth.	Sella normal. Sinuses negative.
DuBE 54	52 Inf.	Loss vision right eye many years ago.	od optic atrophy. os acute retrobulbar neuritis. V od F 2 ft.	Suspicious tonsils. Several carious teeth.	Sella normal. Thickened membrane left antrum. Abscessed teeth.
LH 59	50 Inf.	Blindness right eye within 4 weeks.	on optic atrophy. V nil.	Nose negative. Carious teeth.	Sella normal. Sinuses negative.
MA 7782	38 Inf.	Sudden loss vision left eye following cold 3 years ago.	on optic atrophy. V os 20/200. od 20/30.	Diseased tonsils. Marked nasal obstruction.	Sella normal. Sinuses negative.
GP 7768	50 Inf.	Vision lost 5 years ago following cerebral hemorrhage.	on optic atrophy. V nil.	Deflected septum. Hypertrophied mid. turbinates. Some blocking.	Sella normal. Sinuses negative.
BM 7077	23 Inf.	Gradual loss vision 3 years. Multiple sclerosis?	on optic atrophy. V 20/200.	Deflected septum. Slight hypertrophy mid. turbinates. No blocking.	Sella normal. Sinuses negative.
TM 6242	32 P I	Poor vision since birth.	on optic atrophy. Retinitis pigmentosa. V shadows.	Deflected septum. Slight enlargement both mid. turbinates. Some blocking.	Sella normal. Sinuses negative.
AM 6610	14 P I	Blind since infancy.	on optic atrophy. V od F 8 inches. os light perception.	Normal.	Sella normal. Sinuses negative.

GROUP V.

WAW 51	32 Priv.	Gradual loss vision, first in one eye, then in other.	on optic atrophy. V shadows.	Infected tonsils. Deflected septum, hypertrophied turbinates. Blocking.	Enlarged sella. Sinuses negative.
ST 8149	9 P I	Poor vision since infancy.	on optic atrophy. V F 5 ft.	Hutchinsonian teeth.	Both 6. Some destruction periphery Rt.
NR 8041	10 P I	Gradual loss vision 2 years. Temporal decompression. Headaches relieved.	on optic atrophy secondarily. V od light perception. os shadows.	Small tonsils. No blocking.	Both 7 x 5.
McM J 8522	17 P I	Vision lost following injury when 3 years old.	on optic atrophy. Congenital lues? V nil.	Deflected septum.	Both 6 x 5.5.
BMJ 13321	10 Inf.	Convulsions 9 months ago. Gradual loss vision since.	on optic atrophy. V od 20/10. os F 2 ft.	Nose and throat negative.	Both 7. Eroded at periphery.
SM 6606	76 Inf.	Sudden loss vision 1 year ago, left eye.	os optic atrophy. V od 20/10. os F 2 ft.	Suspicious tonsils. Deflected septum. Enlarged mid. turbinates. Some blocking.	Rt. 6.6 x 5.5. Lt. 7 x 6. Sinuses negative.

RADIOGRAPHS OF

SINUSES AND OPTIC CANALS
Both 5 x 6. Distorted, flattened on top, compressed laterally. (Group II).
Rt. 5 x 6. Lt. 4 x 4.5.
Both 5.5; flattened on top.
Both 5 x 6.
Both 5.5.
Both 5.5.
Both 5.5.
Rt. 5 x 6. Lt. 6 x 6.5.
Rt. 5.5. Lt. 6 x 5.
Rt. 5.5. Lt. 6 x 5; v-shaped.
Both 5.5.
Both 5.5.
Both 6 x 5.
Both 6.
Both 7 x 5.
Both 6 x 5.5.
Both 6 x 5.5.
Both 6 x 5.5.
Both 7. Eroded at periphery.
Rt. 6.6 x 5.5. Lt. 7 x 6.
Sinuses negative.

C.

THE PROBLEM OF MIDDLE EAR MECHANICS.
CHAPTER III. BINAURAL ACUITY FOR AIR
AND BONE TRANSMITTED SOUND UNDER
VARYING CONDITIONS IN THE EX-
TERNAL AUDITORY CANAL.*

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Insofar as the writers are aware, no definite quantitative work has been done on bone transmission acuity. Indeed this type of research was impossible until a bone activating receiver had been devised at the Riverbank Laboratories.¹ It was also considered essential that some idea be gained on minimum binaural audition for air transmitted sound because bone transmission necessarily affects both ears. We reported² that "the sensitivity for binaural minimum audition is consistently higher than that for either ear alone. The amount, however, is not large, and in fact does not lie far beyond the limits of a reasonable error in attention."

*The first two chapters of this series on middle ear mechanics were largely of morphologic character, and were written before the undersigned became associated with the Riverbank Laboratories. Since the outlining of the scope of Chapter III of this series, collaborative research with F. W. Kranz has been made possible through the courtesy of Colonel George Fabyan. The literature cited in the first two articles is appended to this paper and is arranged to correspond with the subject matter of Chapters I and II published in the March and June numbers of the *Annals* for 1922.
A. G. P.

The transmission of sound through the bones of the head to the ear has long been made use of by otologists in the differential tests as to the location of the cause of deafness. Determinations of the length of time a tuning fork is heard, as the vibrations die out when the stem is applied to the head; or of the high frequency limit, as heard with the monochord, are two most common types of bone transmission tests. The first of these two is used in comparison with the air transmission with the same fork in the Rinne test.

The monochord and the tuning fork have hitherto been the principal sources of activation for bone transmission, and while they suffice for certain tests, neither is flexible enough for extended investigations into the mechanism of bone transmission. The lack of control over the intensity has been one of the major difficulties. The tuning fork can do nothing but die out after having been struck, and the intensity of the monochord cannot be varied definitely. The type of instrument required is one in which the intensity can be increased or decreased by known amounts, and held constant at any intensity level desired.

An instrument to meet these requirements has been devised and described. It consists of a modified telephone receiver, which is actuated by the alternating electrical currents from a vacuum tube electrical oscillator. Tones of any frequency desired may be produced by this arrangement. A continuous variation of tone over the range of about one octave may be obtained, and any octave desired may be selected for this type of variation. A more complete description of the electrical oscillator is given elsewhere.³ In the modified telephone receiver, the usual diaphragm was removed and a rather heavy strip or bar of soft iron was put in to span the pole pieces of the receiver, but of course not touching them, being supported at the ends. To this strip or bar a projecting rod with a flat plate is attached. The flat plate is applied to the head or other part to be tested. The requirement for bone transmission is not a large amplitude as in air transmission, but rather a considerable force with comparatively small amplitude; and this is provided by the robust iron strip. The magnetic pull is greater than would be the case with a thin diaphragm. Also the pressing of the

plate against the head does not materially disturb the adjustment or the relation of the bar to the magnets, as would be the case if a thin diaphragm were used, and which would be a serious objection. The intensity of the bone excitation can easily be varied through control of the electrical currents by use of the electrical resistances. With proper arrangement of these resistances the changes produced in the intensity of excitation of the contact plate are known. So we have a source of excitation for bone transmission in which the intensity may be regulated in a known way, or may be held constant at any intensity desired, and in which the pitch is also under complete and minute control. The bone transmission experiments, described below, were conducted with this apparatus.

One of the best known phenomena in the bone transmission for normal hearing people is, if the external auditory canal on one side is closed with the finger or otherwise, vibrations applied to the bone of the head will be referred to the side of the occluded canal. This observation was first made by Wheatstone,⁴ although it is more commonly attributed to Weber,⁵ and is known as the Weber phenomenon of lateralization.

The first experiments were made with the object of determining the factor of increase of bone transmission acuity due to occlusion of the external auditory canals, and to investigate the dependence of this change on the frequency of the tone used and on the individual being tested. The actual values of the intensity applied in bone transmission cannot readily be expressed in absolute units because of the lack of knowledge of conditions at the place of contact. However, a knowledge of the absolute values of intensity is not necessary for determining the ratio of intensities under two conditions or at two different times. The ratio of the intensities at two different times may be regulated and determined by the electrical resistances introduced into the circuit. So the ratio of the low limits of intensity necessary for perception by bone transmission under the two conditions of ears open and ears occluded could be found accurately, the limit of accuracy being determined by the subjective uncertainties in the observations themselves as to whether or not a given intensity was heard.

The alternating current from the oscillator was put through an electrical filter to eliminate the harmonics of the frequency being produced. The current was measured by means of a thermocouple and meter, and then passed through the attenuator. The attenuator consists of several sets of electrical resistances so arranged that any number of these sets may be introduced into the circuit and the resulting attenuation of the current flowing through the apparatus is known. The bone activating receiver is connected to the end of the attenuator.

The procedure of the tests on the effect of occlusion of the external auditory canals on the acuity for bone transmitted sound was as follows: Five frequency ranges were used, each of which covered somewhat less than an octave by a continuous frequency change. That is, the frequency of the tone could be run back and forth between the high and low limits of the range. The five ranges together covered the tones from 256 to about 3300 cycles per second. Each range was tested separately. In any one range the intensity was first reduced by means of the attenuator to a point where none of the tonal range could be heard by the subject, as the frequency was continuously varied up and down. The bone activating receiver was held to the forehead of the subject by himself, and he also had control of the variation of the frequency through the range. Then the intensity was increased and the subject again listened and varied the frequency, determining over what portions of the range, if any, the tone was heard by way of the bone receiver and the forehead. The increases in intensity were continued until all of the frequency range was heard. This was then repeated for each of the other frequency ranges. After this was done with the ears open, it was repeated with the external auditory canals tightly occluded with dental impression compound, which is pliable and can be formed into the cavity when warm but which hardens on cooling. Repeated tests showed that the occlusion by this method was complete, and the adventitious noises and irregularities which are found when the external canals are plugged with the finger were avoided. The changes in the intensity were in a constant ratio of 2.5 to 1, as it was found that changes in smaller steps added nothing to the accuracy because of the limitation imposed by the subjective uncertainty of judgment.

There is much greater uncertainty of judgment as to the absolute limits of audibility in bone transmitted than in air transmitted sounds.

With the current output of the electrical oscillator the same for the open and closed condition of the external auditory canals, the data was taken in terms of the attenuations in the circuit when different parts of the frequency range were heard. The difference in intensity necessary for audition under these two conditions is indicated by the differences in the attenuations present in the circuit and is shown for the four individuals tested in Figs. 1 to 4.

The intensity ratio is shown at the left, and the frequencies of the tones are shown below. The ratios given are those of minimum intensity necessary for hearing by bone transmission with the ears open, to the similar necessary intensity with the external auditory canals occluded; that is, ratios greater than 1 indicate that the bone transmission was improved by closing the external canals, and where the curves fall to ratios below 1 they indicate that for these frequencies the bone transmission was not so good with the ears stopped as with the ears open. The bone transmission reception from the forehead cannot in general be differentiated as between the two ears, so one curve per person suffices for bone transmission.

The data taken are shown by the straight horizontal lines. The ranges used overlapped somewhat and some were repeated, which accounts for the number of lines. As is mentioned above and as is also apparent from the figures, the accuracy of the observations is not very great, but the continuous curve on each figure represents what is considered to be a fair average of the results. The curves from each of these four figures are shown on Fig. 5, together with their general average. This Figure 5 then shows the average change in bone transmission acuity caused by the closure of the external auditory canals for the four individuals tested, and also shows the variations found between these individuals.

The first conclusion to be drawn from a consideration of Figure 5 is that the effect of occlusion on bone transmission acuity is a function of the frequency of the tone used. It is a distinct advantage for all observers at the lower frequencies, but the benefit decreases as the frequency rises until a point

is reached at about 2600 cycles per second, where none of the four individuals are benefited in bone transmission through occlusion of the external auditory canals. The drop in the average curve as the frequency rises is quite regular and gradual.

The wide differences between these normal individuals are also prominent. These variations are so marked that it is obvious that in any individual case it would not be possible to say that he would or would not, or in fact that he should or should not, be benefited in bone transmission by occlusion of the external auditory canals. When only a few subjects are tested the wide individual variations make the average curve quite dependent on what individuals are used, so only the general tendency of the average curve may be considered as representative. It must be borne in mind that this Figure 5 represents only differences in the bone transmission acuity with and without occlusion of the external auditory canals.

The actual sensitivities for bone transmitted sounds with the ears open are shown in Figure 6. The curves were obtained from the amounts of attenuation in the circuit at the limit of audibility. As before explained, these are not expressed in absolute units, and the efficiency of the bone activating receiver used is a large factor in determining the form of the curves. However, the influence of the bone activating receiver characteristics is the same for all of the curves, and so the relative sensitivities of the different individuals are correctly shown. In order to present more clearly the differences between individuals the curves of Figure 6 are redrawn in Figure 7, using the sensitivity of one individual (A) as standard, and expressing the sensitivities of the other three and of the average in terms of their ratios to this one. The ratios are expressed on a logarithmic scale so that equal distances correspond to equal ratios. Thus the ratio of 1 to 2 is of the same importance as the ratio of 5 to 10, and so the distance between 1 and 2 is made the same as the distance between 5 and 10, and the whole scale is on this plan.

It is seen that over a considerable part of the frequency range the bone transmission acuity of observer B is more than ten times that of A, whose bone transmission acuity is here taken as a base for comparison. This means that over a considerable part of the frequency range it takes more than ten

times the intensity for A to hear a tone by bone transmission than it does for B to hear the same tone.

Now the question naturally arises whether or not these wide variations in bone transmission acuity, and also in the effect which the occlusion of the external auditory canal has on bone transmission, are normal variations that might be found in any group of individuals; or whether the variations are connected with differences in acuity for air transmitted sounds. The four individuals tested were all of perfectly normal hearing for conversational purposes. Detailed curves of their acuity for air transmitted sounds, both for right and left ear, are shown in Figures 8, 9, 10 and 11. A comparison of these figures with Figure 7 indicates that good bone transmission is somewhat more closely associated with good air acuity than with poor air acuity, although the connection of bone acuity with air acuity is not at all close in detail.

The determination of the method or route by which bone transmitted vibrations normally affect the end organ is of fundamental importance. The possibilities are not many. (1) The cochlea with its contained fluid may itself be jarred by the vibrations passing through the petrosium to affect the end organ direct (cranial transmission). (2) The vibrations through the bone may affect the stapes footplate through its peripheral ligamentous attachment to the otic capsule, or possibly the membrane of the round window, or both, and thus be transmitted to the fluid of the cochlea in the usual manner of air transmitted sounds. (3) The vibrations may be transmitted to the ossicular chain through the ligamentous attachments to the walls of the middle ear. (4) The transmission to the ossicles may be through the muscle attachments, the *M. tensor tympani* or the *M. stapedius*. (5) The vibrations may affect the drum membrane through its peripheral attachment and be transmitted to the internal ear by way of the ossicles. (6) The vibrations of the walls of the middle ear may give rise to aerial vibrations in the middle ear which affect the oval or round window or the drum membrane or a combination of these. (7) The vibrations of the walls of the external auditory canal may give rise to aerial vibrations which are transmitted by the way of the drum membrane as are the usual aerial sounds from an external source. These routes, or a

combination of them, seem to cover the possibilities. Experimental methods of differentiating between them are being investigated.

One means of identifying the pathway of bone transmitted sounds is by artificially affecting the mechanism of hearing and determining the effect on both bone and air transmission. Perhaps the method by which we would proceed, if we had all of the mechanism of hearing under experimental control, would be to first interfere with the action of the cochlea and determine the effect on bone transmission. If we found that we also interfered with the reception of air transmitted sound we would proceed outwards and restrain or affect in some way the round and the oval windows and note the effect on bone transmitted sounds. Determinations also of the effect on air transmitted sounds for each changed condition would give a criterion of the effectiveness of our restraining or blocking of the auditory pathway.

Experimentally we cannot proceed in this manner; but we can start from the outer end of the auditory path, produce changes in the efficiency as determined by measurements of the effects on air transmitted sounds, and then determine whether or not we have produced a change in the efficacy of bone transmission. If a change is found in bone transmission we may conclude that the route of bone transmitted sounds into the internal ear is at least in part by way of the transmission apparatus affected by the change which was made.

Specifically, if we push the drum membrane inward by increasing the air pressure in the external auditory canal, we can measure the amount of change produced in the reception of air transmitted sounds; and this is a criterion of our disturbance of the mechanism. Now if we find we have produced a similar change in the acuity for bone transmitted sounds by applying this air pressure, we may, of course, conclude that the route for bone transmission is, at least in part, by way of the drum membrane or of the ossicles, the ligaments or the muscles which are affected by pressing the drum membrane inward. If the change in the energy required for air transmission is of the same magnitude as the change in energy required for bone transmission, we may conclude that the affected ele-

ments of the mechanism constitute the major part of the pathway for bone transmitted sounds into the internal ear.

Exactly this kind of an experiment can be and was performed. Interferences in the hearing mechanism were produced by both negative and positive pressures in the air of the external auditory canals. The effect on air transmitted sounds was measured by use of a thermophone sealed in the ear, in connection with the vacuum tube oscillator, the procedure having previously been described, and is similar to that for the bone transmission described above in this paper. The effect on bone transmission was also measured. Tests were made at seven levels of pressure, positive and negative pressures of 10, 20 and 30 cms. of water and neutral pressure, and five frequencies were employed, 128, 256, 512, 1024 and 2048 cycles per second.

Two persons were tested for air transmission to each ear and for bone transmission from the forehead. In the bone transmission tests, the air pressure was applied to both ears at once. The air pressure was applied and also measured by means of a water manometer or U-tube. The bottom of the U-tube was connected to a source of variable water pressure, and one of the upper ends of the tube was connected with the ear, the other end remaining open. The tube leading to the ear contained a stopcock, which was closed after the proper pressure was applied and measured, so that the length of air column connected to the external auditory canal was constant and did not vary with the height of the water column in the manometer. The tube terminations were sealed into the external auditory canals with dental impression compound, and around the edges a layer of liquid glue was placed as an aid in making the cavity air tight. With sufficient care in packing in the wax, the tube could be sealed into the external canal securely enough to hold the maximum positive pressure used. The negative pressures offered no difficulties at all. For the air transmission tests the thermophone was sealed into the pressure tube just at the entrance to the ear.

The results of these tests are shown in Figures 12, 13 and 14. Figure 12 shows the data taken on one individual, subject A of the preceding tests, and the effect of pressure variations on the air transmission acuity for each ear and on the bone

transmission acuity are given. The scale used to show the variations produced is the same sort as used in Figures 1 to 7. It is seen that, in a broad way at least, the effect of pressure changes on bone transmission is similar to the effect on air transmission. In general, the best acuity for both bone and air transmitted sounds is obtained with the normal air pressure on the drum membrane, the acuity decreasing if the pressure is either raised or lowered. A few of these curves do not show a decrease in acuity until the pressure is changed by 20 cms. water. An exception is found in the highest frequency tried, 2,048 cycles per second, for which the acuity for bone transmission did not decrease for the decreases of air pressure but remained constant for 10 and 20 cms. and then showed an increase when the negative pressure of 30 cms. was reached. Undoubtedly related to this increase is an increase in air transmission acuity for each ear at 30 cms. when compared with that at 20 cms. Repetitions of the determinations at this frequency gave similar results. The magnitude of the changes produced by the pressures are about the same for bone transmitted sound as for air transmission with the exception of at 256 cycles per second. A repetition of the bone transmission tests at this frequency showed a variation, with pressure somewhat larger than shown in this figure, although still somewhat smaller than that indicated for air transmission.

Figure 13 shows the results obtained on a second individual, subject B of the preceding tests. For the air transmission of the left ear, the curves are similar to those of Figure 12, but for the right ear and for bone transmission there is a notable difference. The distinctive feature of these last two sets of curves is that the maximum acuity occurs not with normal pressure but with a negative pressure which is, in general, that of about 10 cms. of water, except for the bone transmission at 128 cycles per second, where the acuity is almost independent of the pressure.

The fact that of the four ears tested for air acuity one should show this type of effect is very interesting, but the fact that the bone transmission acuity for this person also shows the same changes is truly significant. This, together with the form of the curves at 2,048 on Figure 12, indicates

very clearly that those members of the auditory system which are affected by the changes in air pressure on the drum membrane are, at least partially, common to the routes of both bone and air transmission to the internal ear. This means that hearing by bone transmission, at least for low intensities, is not wholly accomplished by vibration of the cochlea as a whole, nor by means of the round window, for these would not be affected by the pressure changes made. The pressure changes may, however, have affected the peripheral attachments of the drum membrane, the tension of the ligamentous or muscular attachments of the ossicles, or through the ossicles may have affected the peripheral attachments of the stapes footplate. The experiments are, therefore, the first step toward a definite identification of the route.

It should be here noted that the experiments previously performed and reported by the present writers⁶ as to the effect of pressure on acuity for air transmitted sounds were performed on one ear only, and this was the right ear of individual B, as shown in Figure 13. It was on the basis of the action of this ear that the conclusions were drawn that a negative pressure in the air of the external auditory canal was an aid to acuity. The limitation of the data on which this conclusion was based was specifically noted, and it appears from the present experiments that this particular ear was not representative. None of the other three ears tested showed this effect, and it cannot be said to be representative of human ears in general. In fact, it appears that even for this ear the effect is variable in magnitude. A repetition of the air acuity test on this ear, made about a week later than that shown on Figure 13, showed but small traces of improvement for negative pressure. The results are given in Figure 14. At only three of the frequencies does the maximum acuity seem to be at a negative pressure. The most reasonable explanation of this effect is in the pressure conditions of the middle ear, which may and undoubtedly do vary depending on the functional closure of the auditory tube.

In order to present a somewhat more comprehensive view of the effects of pressure in the air of the external auditory canal for air and bone transmitted sounds, there is shown in Figure 15 average curves for the individuals and conditions combining the five frequencies used.

SUMMARY.

1. The observational error in making bone transmission tests is greater than that found for air acuity. The relatively wide variations in bone acuity are not necessarily dependent on similar variations in the air acuity.

2. Good bone acuity in normal individuals is somewhat more closely associated with good air acuity than with poor air acuity.

3. The advantage to bone sensitivity of occlusion of the external auditory canals seems to be a function of the frequency. The advantage is greatest in the lower frequencies and gradually drops off until at 2,600 cycles per second none of the four observers used was benefited through occlusion.

4. Test on the effects of plus and minus pressures in the air of the external auditory canals on the acuity for air transmitted sound showed the greatest sensitivity when the pressure was atmospheric. Plus and minus pressures decrease the acuity.

5. Plus and minus pressures in the air of the external auditory canals showed that the greatest sensitivity for bone transmitted sounds was found when the pressure was atmospheric.

6. In the one case where the air acuity was greatest at about 10 cms. water pressure minus, the bone acuity was also greatest at this pressure.

7. The evidence submitted shows that the normal route to the labyrinth, for both air and bone transmitted sounds, is through the stapedial footplate.

8. The normal labyrinth is wonderfully insulated against all vibrations passing through the otic capsule, and whether or not this insulation is lifted in cases of otosclerosis through the pathologic changes in the petrosum remains to be investigated.

The Gelle and the Lucae tests are based on the fact that there is normally a decrease in air and bone acuity, respectively, under conditions of plus pressure in the air of the external auditory canal. The explanation for this decreased acuity is based on the assumption that the displacements of the drum membrane, due to plus pressure, increases the pressure within

the labyrinth through the agency of the ossicles on the stapedial footplate. Our results, however, show that not only a plus pressure but also a negative pressure in the air of the external auditory canal decreases both bone and air acuity for normal hearing people. Either we must assume that negative pressures in the labyrinth produce similar effects to positive pressures or we must seek some factor outside of the assumed changes within the labyrinth to account for this phenomenon. Kato⁷ has shown that medial displacement of the drum membrane does not produce an increase in the labyrinthine pressure so long as the intrinsic muscles are intact. It seems reasonable, therefore, that the factor affecting acuity under pressure lies outside the labyrinth and is the same sort of factor for both positive and negative pressures in the air of the external auditory canal.

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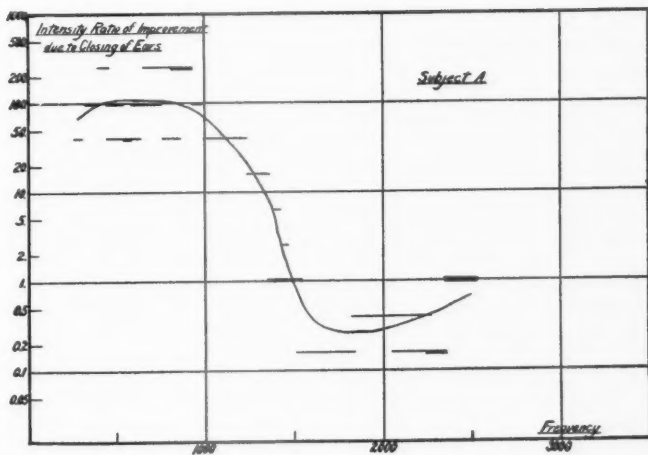


Fig. 1.

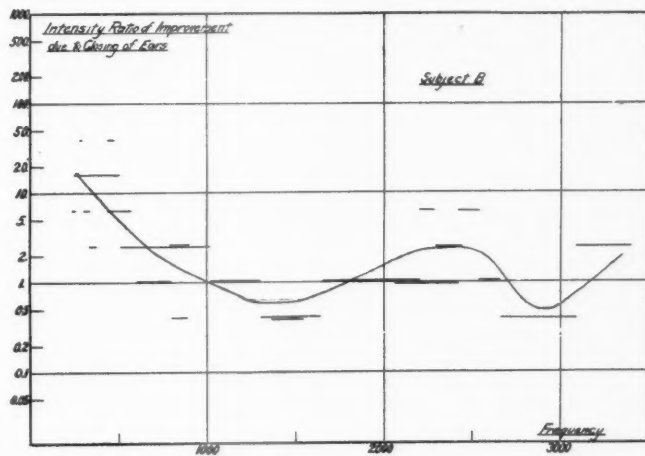


Fig. 2.

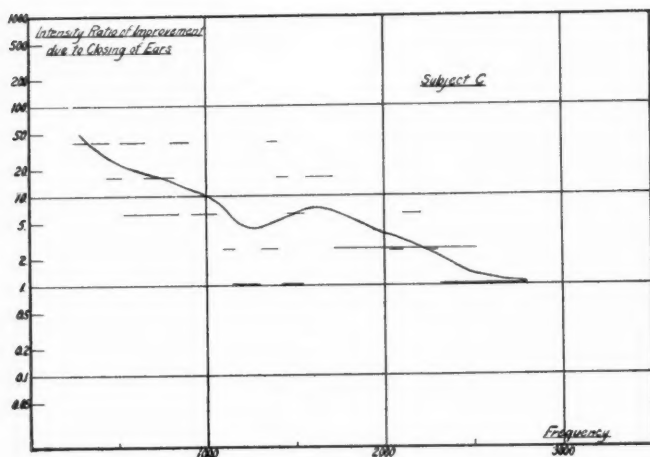


Fig. 3.

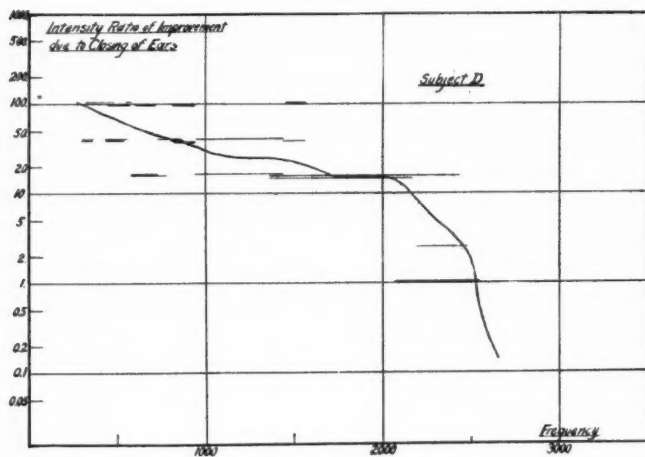


Fig. 4.

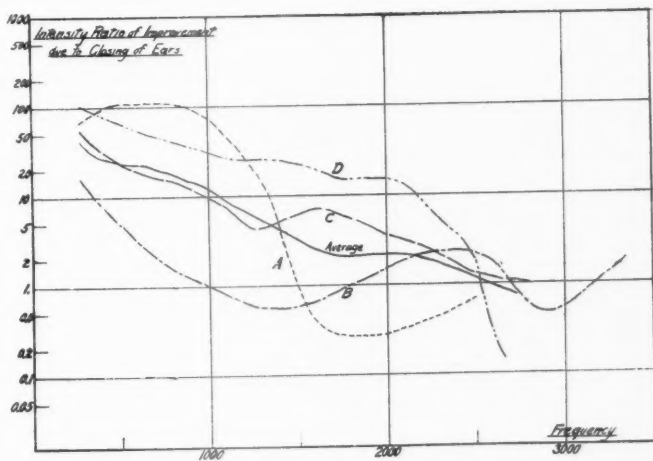


Fig. 5.

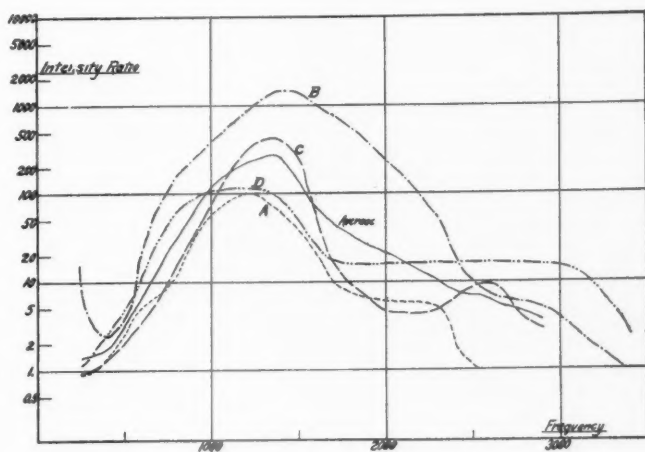


Fig. 6.

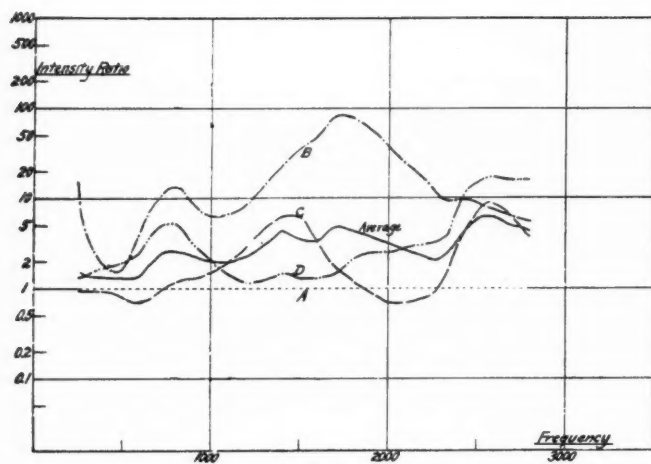


Fig. 7.

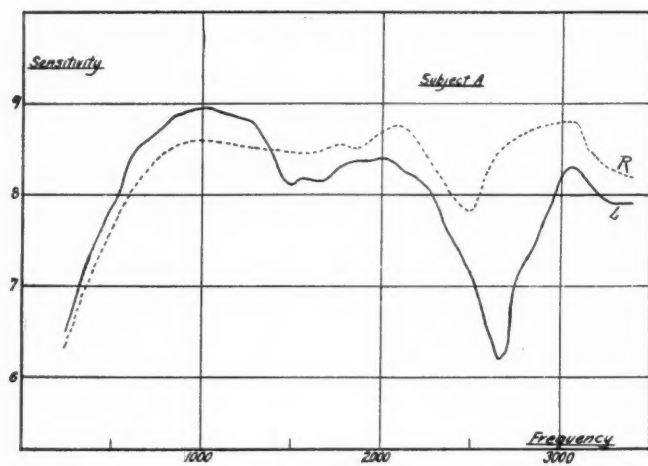


Fig. 8.

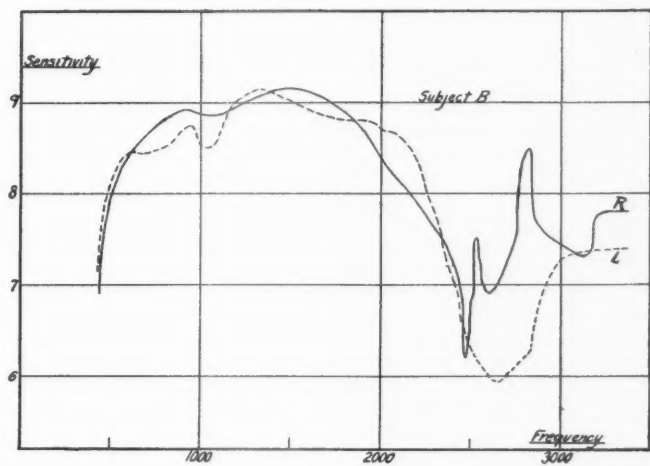


Fig. 9.

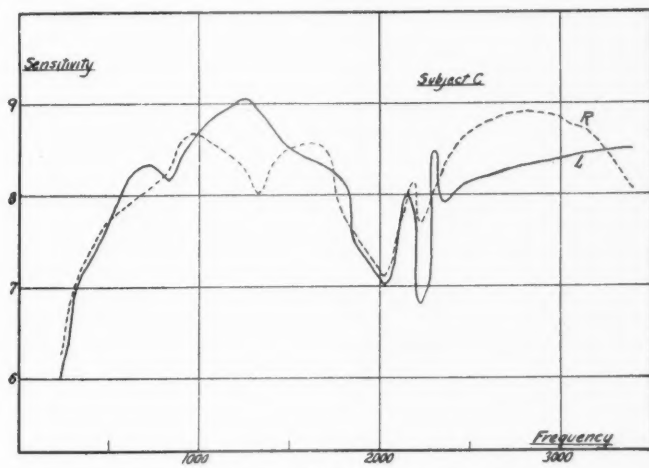


Fig. 10.

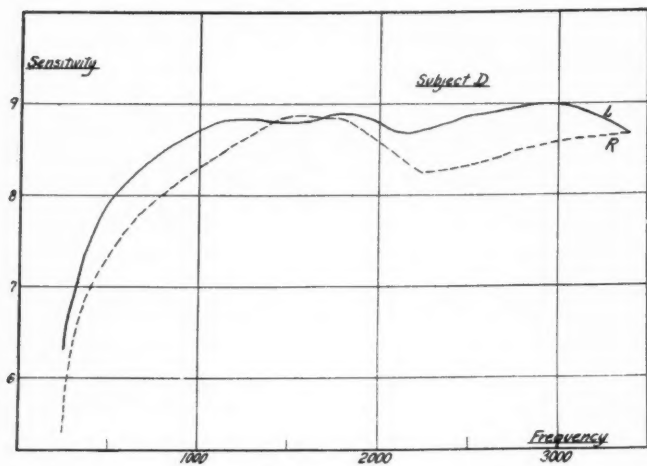


Fig. 11.

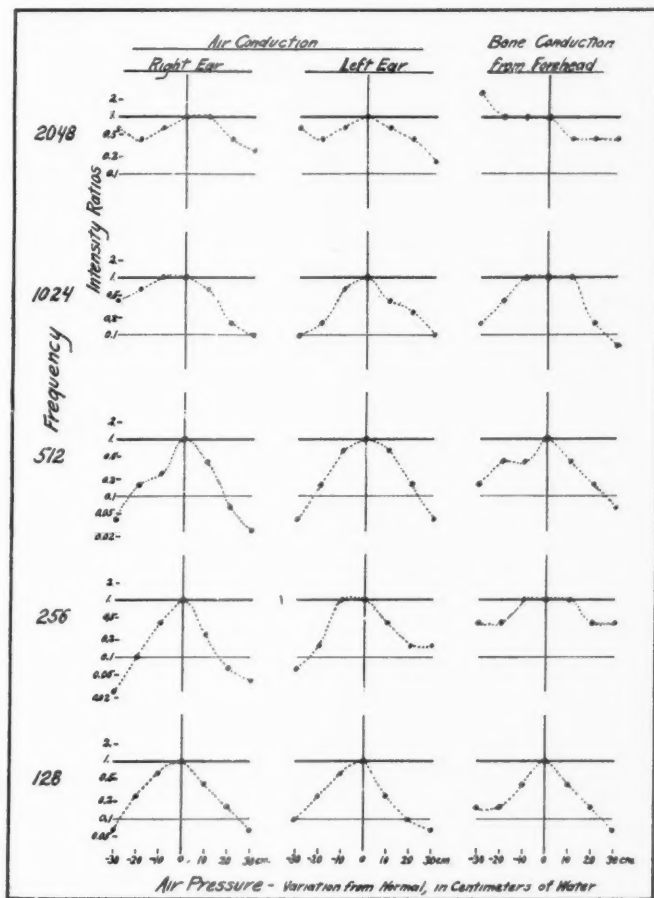


Fig. 12.

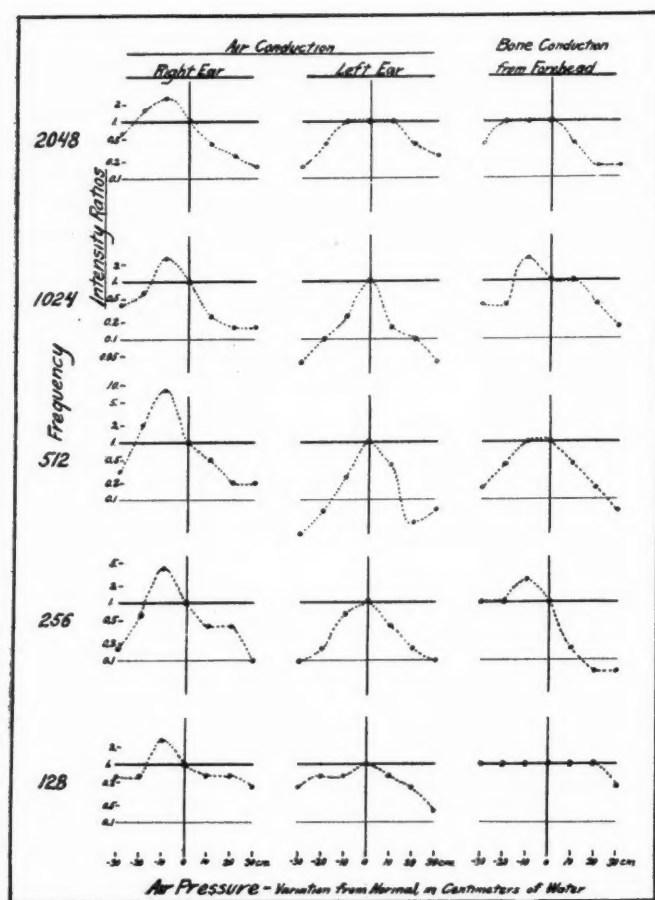


Fig. 13.

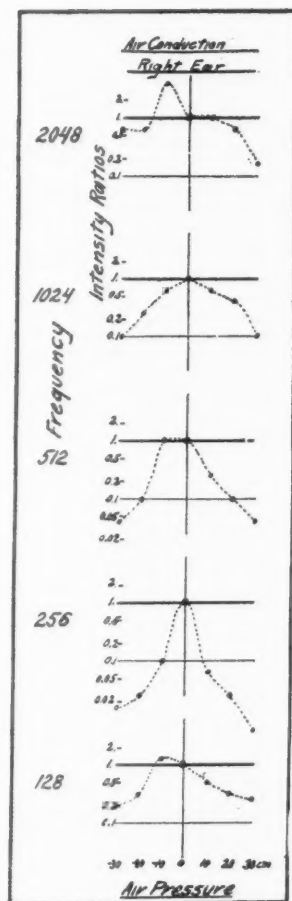


Fig. 14.

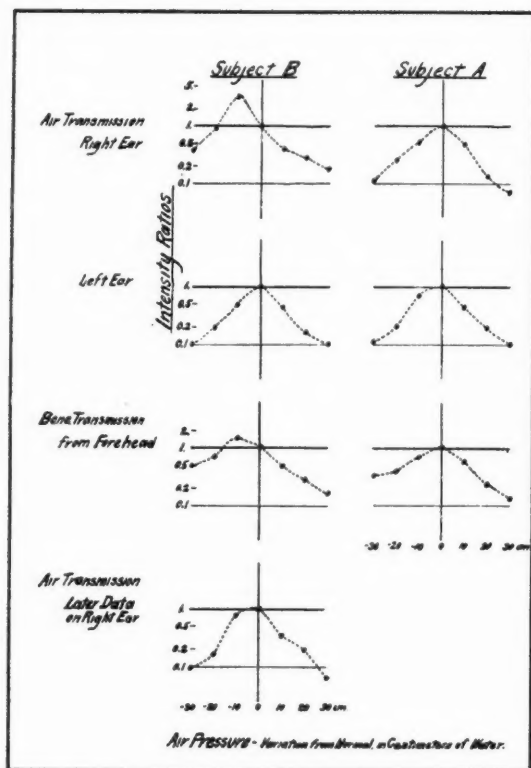


Fig. 15.

CI.

THE CHOLESTEATOMA OF THE MIDDLE EAR— ITS ETIOLOGY, PATHOGENESIS, DIAGNOSIS AND THERAPY.

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Of all the middle ear suppurations which are bound to claim the interest of ear specialists, cholesteatoma occupies the first place, because, according to the very best statistics, we must reckon with a mortality of $1\frac{1}{3}$ per cent.

My teachers, Bezold and Siebenmann, through their own work and that of their pupils, have cleared up the question of cholesteatoma in its every aspect.

The statistical researches by Scheibe and Schlittler cover the entire material from the Munich and Bâle clinics for 20 years of work by these two scientific observers. The material from Munich dates from 1881 to 1901, the one from Bâle from 1898 to 1917, and embraces about 83,000 patients, with 131 fatal cases. The latter were carefully examined macroscopically and also microscopically, at the clinic of Siebenmann. These investigations resulted in a series of conclusions to which I always refer in my clinic and which I should like to present to you.

It is unnecessary to discuss in detail the definition of aural cholesteatoma. We mean by this term a form of chronic middle ear suppuration, with its results, which is characterized by an epitympanic and marginal perforation, by a continuous fetid discharge, and especially by the epidermization of the mucous membrane in the middle ear.

The importance of cholesteatoma is due to its frequency. Among the 83,000 patients of the Munich and Bâle clinics there were in all 12,000, 14.5 per cent, of chronic middle ear suppurations and in one-third of these cases cholesteatoma was a complication. There is, therefore, an average of one cholesteatoma patient to three chronic middle ear suppurations.

The mortality in case of cholesteatoma, however, differs. Bezold's statistics give us a mortality of $3\frac{1}{3}$ per cent, Siebenmann's only $1\frac{1}{4}$ per cent. Of the 131 cases of death in these two clinics, 50 can surely be traced to cholesteatoma. This disease is, therefore, the leading lethal ear affection.

The simple chronic middle ear suppuration can be called almost harmless in comparison, since, at best, only one fatal case could be attributed to it. Lately similar statistics were published by the clinic of Scheibe (Erlangen), confirming in every respect the two extensive statistical findings cited. The mortality amounted to $1\frac{1}{3}$ per cent, though the period of time was somewhat shorter and the number of patients also smaller.

Opinions today still differ about the origin of cholesteatoma. In the literature we find various descriptions of cases of congenital cholesteatoma, but neither in Munich, Bâle nor Zürich has such a case ever been observed among patients numbering about 100,000. The tests which formerly were accepted as proofs of the congenital character of the cholesteatoma, for example, the existence or absence of elastic fibers in the cholesteatoma matrix, were not confirmed. It would, therefore, be very difficult to prove that a cholesteatoma is really congenital, and at any rate such an observation would be of such a rarity as to lose all practical importance for diagnosis or for therapy.

It seems certain, therefore, that cholesteatoma is acquired, with its origin during life. There is even a difference of opinion of the cause of the squamous epithelium in the middle ear, which is characteristic for cholesteatoma.

The opinion of Habermann and Bezold, who claim that the epidermis grows from the external meatus into the middle ear, has the greatest number of adherents and is applicable to most cases. The second assumption regarding the metaplasia (Wendt) has fewer followers. For the better understanding of the Bezold-Habermann theory it should be pointed out that the mucous membrane of the tympanum, especially in its higher parts, consists of a flat and frail epithelium, which originally was ciliated, as is usually seen in the neighborhood of the eustachian orifice. Under the influence of inflammation, however, this low or flat epithelium betrays its character as

mucous membrane, because it becomes cylindric and so reminds us of its origin.

The histogenetic process of cholesteatoma formation is most easily observed in cases of scarlatinal otitis. The middle ear affections in the course of this disease run an absolutely analogous course with those of the nose or tonsils, where we are able to observe them much more easily, especially their healing by epidermization from the margins. A similar process will take place in the middle ear, though somewhat modified by local conditions. Owing to the necrosis of the inflamed mucous membrane, which also affects the drumhead, ulcerations arise in the tympanum. The fibroid false membranes can be seen by otoscopy. Often even the bone of the mastoid becomes necrotic, as well as the mucous membrane, and leads to an empyema, which, when operated on, discloses large bone defects or even sequestra.

The healing of these necrotic middle ear inflammations begins by the exudation of the false membranes and the production of purulent discharge. The formation of epithelium cannot proceed from the cylindric layer, because, as is well known, this kind of epithelium does not possess any regenerating quality. The squamous epithelium of the meatus, with its strong growing tendency, will push its way far more easily over the bared margo into the middle ear.

We find similar conditions in the nose after long suppurations, where the mucous membrane becomes overgrown and is replaced by the epidermis. There can be no doubt about the fact that this epidermization after middle ear suppuration represents a healing process, because we observe that the discharge decreases more and more as the formation of skin increases. But the squamous epithelium of the meatus can only grow into the middle ear if the perforation of the drumhead reaches as far as the margo, especially its upper part. Therefore, the necrosis of the mucous membrane and an epitympanic perforation play the most prominent part. No cholesteatoma can arise with a central perforation. The proof of the correctness of this fact was found by Ulrich in the circumstance that of 458 cases operated on for cholesteatoma in the clinic at Bâle 98 per cent presented an epitympanic but never a central perforation.

Bezold and Habermann brought the first histologic proofs of the ingrowth of epidermis into the middle ear, and since then their observations have been always confirmed. In very rare cases the epithelium may enter the middle ear by the help of a fistula in the meatus, when there are large cavities near the antrum, as observed after scarlet fever, etc.

In its pathogenic aspect the cholesteatoma in cases of isolated perforation of the membrana Shrapnelli assumes a place of its own, because middle ear suppurations never perforate at this spot and cannot be held responsible for the origin of cholesteatoma.

Bezold and other authors have noticed that the changes in the membrana Shrapnelli are mostly bilateral and similar. Owing to the absence of a fibrous layer, the membrana Shrapnelli will be easily pushed inwards by unequal pneumatic pressure upon its surface.

As a sign of acute and chronic obstruction of the tube, we observe a retraction of the pars flaccida, which finally, stretched out, tent- or cone-like, reaches the wall behind and occasionally tears, and through this perforation the ingrowth of epithelium again becomes possible. Bezold assigns an etiologic rôle to eustachian obstruction in consequence of persisting adenoids.

According to the latest research work of Lange, Hellmann, it appears that under the influence of chronic irritations, following eustachian catarrh, the epidermis of the pars flaccida shows a tendency to deep ingrowth, forming conelike extensions which expand in different directions. They push the mucous membrane of the middle ear before them, or force it from its base (Hellmann) and produce epidermic scales in the center.

This furnishes the explanation for the origin of cholesteatoma, even in cases of intact, unperforated but retracted membrana Shrapnelli. Something similar is known to occur in the formation of epidermic pearls on the outside of the drumhead.

If in this way we may regard the histogenesis of cholesteatoma as having been cleared up, it is now of importance to know the diseases which lead to initial and primary suppura-

tion of the middle ear, with the resulting formation of cholesteatoma.

Ulrich formed the following conclusions from his study of the histories of 458 cases operated on at Bâle:

In more than 33 per cent the original middle ear suppuration appeared in the course of exanthematous infectious diseases, scarlet fever, measles, diphtheria, typhoid fever, erysipelas. This is readily explainable, because we know that the affections of other mucous membranes (nose, throat, etc.) often show a necrotic character and superficial ulcerations, which in the tympanum form the basis for the cholesteatoma.

While the development of cholesteatoma generally covers a period of many years, in some individual cases, especially of scarlet otitis, it may be observed to take place within a few months.

The different phases, acute necrotic otitis, melting away of the ear drum, mastoiditis with empyema, antrum operation, epidermization of the wound cavity and radical operation, follow each other at short intervals. I recall four cases from my days at Bâle which required the radical operation for cholesteatoma only a few months after the original scarlatinal otitis.

Considerable importance must also be attached to the ear suppurations after influenza and similar affections of the upper air passages (about 20 per cent).

But in almost half of the cases investigation into the histories of the patients did not show any distinct causal affection, the discharge having either started during infancy without any important symptoms or remaining quite unnoticed.

A painstaking study of the Bâle material resulted in the valuable statement that the relationship between tuberculosis and cholesteatoma, to which Schwartz had already referred, was confirmed quite conclusively.

That middle ear tuberculosis heals by the formation of cholesteatoma has already been described by Hegetschwiler at Bezold's clinic, since the histologic changes of tuberculosis form an especially favorable soil for epidermization or cholesteatoma formation.

Ulrich could, in 42 per cent, prove a florid tuberculosis or an already existing tubercular affection, or at least a very definite hereditary disposition. The percentage of tuberculosis was especially large among those cases of cholesteatoma where a causal affection of ear trouble could not be given with any certainty.

The lack of symptoms in cases of tuberculosis of the middle ear is generally known. Therefore in the future a very prominent part has to be assigned to tuberculosis of the middle ear and to tuberculosis in general, in connection with the origin of cholesteatoma.

Though cholesteatoma, as a rule, has to be considered a healing process, it involves great dangers because of two factors, namely, accumulation and putrid decomposition of the epidermic scales. In consequence of the irregularity of the tympanum, the epidermization also extends into smaller recesses, which soon get filled up by exfoliated epidermic masses. Like a little growing ball or a sebaceous cyst (atheroma) of the scalp, the cavity filled with concentrically arranged cholesteatoma masses gets larger by pressure on the surrounding structures. The growth takes place at the cost of its environment; the bone wastes away, the ossicles get corroded (falsely designated as caries), the labyrinthine capsule or the lamina vitrea of the middle and posterior cranial fossæ are destroyed. In moisture and warmth the epidermic scales form an excellent breeding place for putrid decomposition, thus causing an inflamed cavity, which lies side by side with labyrinth and dura or sinus, and involves the danger of spreading to vital organs. Moreover, through granulation tissue formations (here called polypi), the conditions for drainage become still more unfavorable and the retention more imminent, so that the deleterious effect of the cholesteatoma is plainly evident.

Diagnosis: From what has been said already, it will be seen that the diagnosis of aural cholesteatoma is today much easier. In every chronic middle ear suppuration with fetid secretion and epitympanic marginal perforation, cholesteatoma must be suspected. The exact localization of the drumhead perforation is ascertained with a hook probe and magnifying glass. Existing granulations and polypi, which always indicate an intense

necrotic inflammation, are removed, causing the drumhead to become more distinctly visible.

Intratympanic syringing with attic tubes discloses in many cases epidermic scales, which make the diagnosis of cholesteatoma absolutely certain. The microscopic examination of squamous epithelium and cholesterin crystals is interesting but unnecessary for our purposes. The pneumatization of the mastoid can be examined with X-rays, but no method can to any extent take the place of exact otoscopy with probe, magnifying glass and intratympanic syringing.

The greatest importance should, therefore, be attached, when teaching otoscopy, to the distinction between epitympanic and central perforations. This fact should be made known even to the general practitioner, as the first symptoms of glaucoma or of strangulated hernia.

The therapy of aural cholesteatoma is also much simpler and clearer in modern practice. We must keep in mind the following facts:

A complete *restitutio ad integrum* is not possible, on account of the destruction of mucous membrane and bone, but since the danger of cholesteatoma lies in the accumulation and putrid decay of cholesteatoma masses, it must be our aim to secure a dry cholesteatoma cavity. This cavity should be broadly connected with the meatus and tympanum, so that the desquamation of the epithelial scales may not result in accumulation. Nature has shown us the way, namely, in spontaneously breaking through towards the outside or the meatus, the so-called spontaneous radical operation.

Of course, the treatment must be carried out by the surgeon—home syringing is not only without effect but even dangerous. Small cholesteatoma cavities with good conditions for discharge—that is, with a large opening into the tympanum or meatus—may be treated conservatively. With the help of the attic tubes of Hartmann, they can be thoroughly cleaned of cholesteatoma masses, well dried and disinfected with boracic acid powder in such a manner that neither accumulation nor putrid decomposition will occur.

In most cases (about two-thirds) this treatment will be sufficient and very gratifying if applied with the necessary

patience, persistency and adequate technic. It must be continued until the suppuration entirely stops. Secretion often ceases surprisingly quickly and may not reappear for years. The intelligent understanding of the patient, however, is a prerequisite for success, and especially for further prognosis. He must return for treatment as soon as there is the slightest relapse. A regular supervision within various intervals is urgently required.

But where the cholesteatoma cavity is large or the opening to the tympanum narrow—for instance, in Shrapnell cholesteatoma—the dangers mentioned above are existent, and the treatment must be modified accordingly. Cleansing with attic tubes is to be tried in all cases, except when there are signs of a complication or acute inflammation. When this does not bring the desired result, one should resort to the radical operation. Constant fetid discharge and desquamation of cholesteatoma masses indicate that the cavity cannot be completely cleansed or freed from the epithelial scales. Therefore, the cavity must be laid bare by operation and connected broadly with the outside.

I do not need to describe here the technic of the radical operation. I will only mention that in accordance with the method of Siebenmann, we make it a principle to be careful of the cholesteatoma matrix. We do not remove it by curetment, for it cannot be replaced with anything better, not to speak of the danger of contact with the stapes and the promontory wall. A complete survey of the cavity is provided by the meatal plastic, so that it may be cleaned out and controlled, even by the house physician.

The indications for the radical operation of cholesteatoma, according to the best judgment, are provided by the following conditions:

1. Inflammation of the cholesteatoma with threatening symptoms (pain, rise of temperature, symptoms of labyrinth, dura or sinus complications).
2. Failure of the conservative treatment in spite of a consistent application during four to six weeks.
3. Constantly recurring suppuration in connection with the cholesteatoma, even when it can be stopped periodically by conservative treatment.

4. Insufficient cooperation of the patient or the impossibility of an exact conservative treatment, owing to general conditions, such as lack of ear specialists, distant places of residence, etc.

The extraction of the ossicles or exposure of the cholesteatoma cavity, starting from the meatus, need not be mentioned here. Undoubtedly such minor operations will be successful in some cases, but a survey and exposure of an irregular cholesteatoma cavity will not be possible by these partial operations; therefore relapses will be far more frequent.

The radical operation is absolutely contraindicated in cases of chronic middle ear suppuration with central perforation and without cholesteatoma, because the operation endangers the hearing and, moreover, does not do away with the discharge.

The prognosis of aural cholesteatoma depends in the first instance on the timeliness of the diagnosis.

The diagnosis having been made, and the patient as well as the physician, being fully advised regarding the importance of the complaint, the dreaded complications should be more and more reduced.

Proofs of this are furnished by statistics from places, with physicians of good professional training, where a constant decrease of the fatal complications has been noted. Thus, for instance, in the years 1881 to 1907, the number of deaths noted by Bezold still amounted to $3\frac{1}{3}$ per cent, while Siebenmann noted only 1.24 per cent during the period from 1896 to 1917. The increasing advancement of otologic surgery, especially since the invention of the radical operation, by Zaufal and Stacke in 1890, is manifested significantly by the decrease of deaths.

With the help of an early diagnosis, the prognosis for the hearing has also considerably improved, because through conservative therapy and a carefully executed radical operation, the hearing has generally been preserved and in no few cases has been improved.

The care of every kind of ear disease, especially during childhood, will assist in the prophylaxis of cholesteatoma. This includes the treatment of diseased tonsils and adenoids. It is, therefore, advisable that all children as well as fever hospitals

should have well trained specialists at their disposal, because nonspecialists have considerable difficulty with the diagnosis of children's ear diseases on account of the anatomic conditions. At the same time all ear specialists should take part in the fight against tuberculosis, since we must recognize what a part this disease plays, even in our apparently remote special domain.

In conclusion, I wish to express the hope that I have been able to demonstrate how dry statistics and laboratory studies in this field have helped us to form important conclusions, and also how important an exchange of common experiences must be for the benefit of our patients who suffer from aural disease.

CII.

ORBITAL CELLULITIS COMPLICATING PARANASAL SINUS DISEASE IN CHILDREN. REPORT OF CASES.

By T. R. GITTINS, M. D.,
SIOUX CITY, IOWA.

The quite frequent occurrence of paranasal sinusitis in infants and young children here in the Middle West is a matter, I think, of common knowledge. Dean¹ in the past four or five years, with a wealth of material, has covered the subject from many angles.

These three cases with orbital complications are reported for two reasons:

1. To add a little clinical evidence to the fact that orbital cellulitis does appear in infants and children as a complication of sinus diseases. Phelps² in an article reports ten cases with an excellent review of the entire subject. Coming from Minnesota, this last group of cases emphasizes again the prevalence of sinus infection in the children of the Mississippi Valley region.

2. To urge intranasal drainage of the sinuses, first, in the treatment of these orbital complications. In the three cases reported in this paper, as well as in several cases in adults, intranasal drainage of the sinuses was the only operative procedure. In cases 1 and 2 there was no after treatment following the original irrigation of the sinuses. It is the practice of my associate, Dr. Naftzger, and myself to drain the sinuses intranasally first and then watch the orbital condition. If the cellulitis does not subside or pus is deemed present in the orbit, external drainage is then done. Our records show that the last six cases of orbital cellulitis, three of them reported here and three adults not reported, cleared up with intranasal drainage of the involved sinuses and no external incision in the orbital tissues.

Case 1.—R. W., December 23, 1923, age 20 months. Four days ago the eyelids on the right side began to swell. This

edema has gradually increased and exophthalmos has developed. There has been no pus in the eye, no history of injury or insect bite. The child has been having a bad cold in the head for the past six weeks, with considerable nasal discharge and tearing of right eye. For the past two or three days the child has been restless, has not been eating or sleeping well and cries a great deal. Tonsils and adenoids were removed six months ago.

Examination: Very marked edema of both eyelids, right, and considerable exophthalmos; limitation of movement of right eyeball in all directions. Conjunctival injection, bulbar and palpebral, but no pus in eye. Temperature, 102° ; white blood count, 21,500. Radiograms of sinuses were taken under ether anesthesia and showed both antra and ethmoid cells but no sphenoid or frontals evident. Left antrum was clearly outlined but on the right side there was very poor outline of the roof of the antrum cavity, giving the impression of a lack of bone in this region. Tonsils and adenoids were removed four months ago. The right antrum was punctured just above anterior end of right inferior turbinate. Some thin seropus obtained on irrigation. Opening enlarged with a small rasp. Exploration with blunt probe proved that roof of antrum lacked bone. No necrotic ethmoid cells were found. On the following morning there was much less edema of eyelids. The patient slept well during the night and was able to open the eye. Temperature, 100° . Two days following the irrigation of the antrum the edema had practically disappeared from the eyelids and the exophthalmos had receded. There was rapid improvement each day.

On December 28, five days after entrance to the hospital, the patient was discharged. Temperature was normal, white blood count 12,000, exophthalmos had disappeared, as also had the edema of the lids. There was no treatment of the sinuses following the first irrigation. The child, who has been under observation since, has had no recurrence of the orbital cellulitis and the general condition is very good. There has been some discharge from the nose at times but no severe sinus symptoms.

Case 2.—J. B., Jr., February 3, 1922, age 3 years. About four days ago eyelids on right side began to swell. Swelling

gradually increased, eyeball became inflamed and there was some exophthalmos, which gradually became worse. The child had been feeling all right before the swelling of lids began. There was slight cold in head but very little discharge from the nose.

Examination: Very marked swelling of both eyelids, right. Very definite exophthalmos, the eye being practically immobile. There is considerable conjunctival injection, bulbar and palpebral, but no pus. Temperature 101° , pulse 140, respiration 24, white blood count 22,350. Radiograms of sinuses shows both antra blurred, no frontals evident. Ethmoid cells present and apparently blurred. Under ether anesthesia the right antrum was irrigated; thick mucopus was found. The anterior ethmoids right side were quite necrotic. The opening into the antrum under the inferior turbinate was enlarged slightly with a rasp, and anterior ethmoid cells were curetted without removing any middle turbinate. Adenoids were also removed. On the following day edema of eyelids subsided very much, and there was less exophthalmos. Temperature was 99° , white blood count still high, 25,900.

There was rapid reduction of orbital cellulitis, and at the end of eight days after operation the eye appeared normal. There was no further treatment of the sinuses after the first irrigation. At the present time, over two years following the operation, the child is still under observation and has had no further trouble with the eye or sinuses.

Case 3.—S. P., age 12 years, came to the hospital January 21, 1924, with a typical picture of orbital cellulitis, right. There was a history of discharge from nose, fever and headache, with rapidly increasing edema of eyelids and exophthalmos right side. Radiogram of sinuses showed marked involvement of right antrum, ethmoids and frontal. Temperature 100° , white blood count 18,600.

Intranasal drainage of the antrum, anterior ethmoids curetted and nasofrontal duct enlarged. There was much thick pus in the antrum. Anterior ethmoids were very necrotic.

On the next day there was much less edema of eyelids and less exophthalmos.

There was a rapid improvement. The patient was dismissed from hospital January 24th, fourth day after operation; practi-

cally all edema of lids and exophthalmos had disappeared, temperature and white blood count normal. The antrum was irrigated daily for the first five days, but no irrigations since. No recurrence of trouble.

REMARKS.

1. In case 1, a child 20 months old, a marked orbital cellulitis subsided rapidly after irrigation of the antrum on that side. The X-ray picture suggested a dehiscence in the bony orbital wall of the antrum. This was confirmed by the probe at operation. There was no after treatment.

2. In case 2, a child of three years, an orbital cellulitis subsided rapidly with but one irrigation of the antrum and a curetment of the anterior ethmoid cells. There was no after treatment.

3. In six consecutive cases of orbital cellulitis, three in children and three in adults, there was rapid and complete recovery following intranasal discharge of the sinuses, with no external incision in the orbital tissues.

4. We look upon inserting a drain into the orbital tissues as do the general surgeons when the knee joint or brain are involved. We feel that every effort should be made to reduce the orbital cellulitis without drainage of the orbital tissues themselves. If pus is actually present in the orbit at the time of operation or develops later, then, of course, external drainage is indicated.

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CIII.

DR. HENRI LUC.

By RALPH A. FENTON, M. D.,

PORTLAND.

Dr. Henri Luc, Officer of the Legion of Honor, died at his home in Paris, France, September 25, 1925, at the age of 70, and was buried at his birthplace in St. Omer.

Elder English-speaking otolaryngologists who were privileged to know him will recall the erect figure, the alert eye, the bushy red beard of this great Norman, whose last visit to the United States occurred at the Boston meeting of 1912. Meticulously accurate in English speech, as well as in German, Spanish and Italian, Professor Luc was one of the important agencies of the quarter century before the war toward an interchange of scientific information between the clinical chiefs of various European countries and of America.

It is regrettable that so few Frenchmen have followed Luc's example regarding other tongues.

Numerous Americans of prominence were his patients in Paris, either at his old home in the aristocratic rue de Varenne, in the St. Germain quarter, or at his clinic in the rue de Chateaillon. It was a privilege to the visiting otolaryngologist to be invited into either clinic, where other confreres from Japan to Buenos Aires might be encountered.

Luc's ideas regarding the middle ear, mastoid and the ethmoid, as stated in his "Lecons sur les suppurations de l'oreille moyenne" (1900), were far in advance of his time, and did much to bring French thought abreast of the work which was being developed in American, German and Austrian clinics. His name is attached to important surgical procedures on the antrum, the frontal and the ethmoid, and various instruments of his devising are in daily use. His pupils have been active and progressive men, especially in France, Spain and South America.

The war of 1914-1918 saddened Professor Luc, for it brought the collapse of many international scientific relationships and friendships. Though far above military age he worked hard and well in the war surgery of the Paris clinics. With Morestin at the Val-de-Grace, and elsewhere, he did much work in the repair of war injuries. Increasing disability, inability to sleep because of pain, the death and suffering of many old friends, including Morestin, embittered his latter years, and he spent much time in the South of France at various resorts.

He was always glad to see American friends, and was most grateful for America's aid to his stricken country. He is survived by Mme. Luc, by two married daughters, and by a son twelve years of age.

He was a corresponding fellow of the American Laryngological Association since 1897, an honorary member of the American Otological Society since 1904, and of the American Laryngological, Rhinological and Otological Society since 1896.

SOCIETY PROCEEDINGS.

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL
SOCIETY.

Meeting of Monday, April 6, 1925.

THE PRESIDENT, DR. E. P. NORCROSS, IN THE CHAIR.

CLINICAL PROGRAM.

Laryngotomy and Laryngectomy Case Exhibition.

BY DR. JOSEPH C. BECK AND DR. HARRY L. POLLOCK.

DR. BECK: We have several cases of laryngotomy and laryngectomy that have been shown before. They are presented now because they have remained free from recurrences **and also because a voice has been added by the use of an instrument perfected by Dr. MacKenty.**

Case 1.—Mr. F., first seen in October, 1921, aged 62, with complaint of difficulty in breathing and hoarseness of two years' duration. In June, 1920, a thyrotomy was performed at another clinic and the growth was removed, which was proven to be carcinomatous. There was a recurrence soon afterward, and in May, 1921, he returned to his laryngologist and was informed that further surgical measures should not be attempted, but that radium and X-ray should be employed. At our first examination we found a carcinoma growing through the thyroid incision and extending externally. There was so much difficulty in breathing that an immediate tracheotomy was performed. On November 9, 1921, under synergistic analgesia, a laryngectomy was performed. He had a stormy convalescence complicated by two severe attacks of erysipelas. But with this exception, he made a speedy recovery. In February, 1922, he was discharged from the hospital, having gained about forty pounds. Up to the present time there has been no evidence of recurrence. This instrument (MacKenty) the patient has had but a short time and is unable to use it very satisfactorily. However, at times he can make himself clearly understood and probably with practice he will have a good voice.

Case 2.—Mr. S. was first seen on February 12, 1925, with history of hoarseness of four months' duration. On January 16th he had his tonsils removed. Our examination revealed an irregular mass on the left cord, with some whitish deposits. Direct suspension was somewhat difficult, and a specimen obtained by this method was reported by the pathologist to be fibropapilloma with no evidence of malignancy. On account of this finding a laryngotomy was done under synergistic analgesia on March 11th, the growth thoroughly excised and the wound closed. This specimen, which was sent to the laboratory, was returned with the report of "squamous cell carcinoma." On March 19th a laryngectomy was performed, but the larynx had to be removed in two pieces owing to the previous laryngotomy. The man is in very good condition and receives his nourishment through the duodenal tube which is carried through his nose.

Case 3.—Mr. H., 70 years of age, was first seen on July 22, 1921, with a history of hoarseness of eleven months' duration. Examination revealed a growth on the right vocal cord. On July 26th a specimen was removed by suspension laryngoscopy, and the pathologic report of squamous cell carcinoma was made. The patient received some radium treatment, also a course of deep X-ray therapy, which apparently made no impression on the carcinoma. On September 15, 1921, under synergistic analgesia, a laryngotomy was performed and the growth excised. The incision was closed primarily and the patient made an uneventful recovery. It is now about three and one-half years since the laryngotomy was done and, as you see, the patient is in good physical condition, and examination shows no recurrence of the growth.

Case 4.—Mr. B., aged 58, was operated on by Dr. Lynch in 1924, and as he is a resident of Chicago, was sent to us for observation. He is in excellent physical condition and at the present time is free from any recurrence. He has a Mac-Kenty apparatus, but has had it only a very short time and is inexperienced in its use, but at times has a very distinct voice.

Case 5.—Mr. R., aged 70, was first seen by us in 1911. He gave a history of hoarseness, with an operation performed four months previously by a Dr. Rhodes of this city, who operated

by the indirect route and removed the growth. At our first examination a recurrence was found, and a short time afterwards a laryngotomy was done, the growth removed, with immediate closure of the external wound. During the first forty-eight hours he experienced quite a little difficulty in swallowing and breathing, but this subsided without any further surgical interference, and since that time, as far as the larynx is concerned, he has been perfectly well, with no evidence of recurrence. The pathologic specimen showed an adenocarcinoma with a great deal of connective tissue spread through it. He was examined again during the past week and is absolutely free from any recurrence.

Case 6.—I simply desire to report this case, as the man is still in the hospital and did not feel like coming out tonight. He is 75 years of age, was operated on three months ago following a microscopic examination of the specimen, which proved to be a squamous cell carcinoma. The usual laryngectomy was performed under synergistic analgesia. He has had two complications: First, an osteomyelitis of the two upper ribs, which resulted in large abscesses with final sequestration of the ribs; second, a paroxysmal tachycardia, which at times became alarming. These attacks later came at less frequent intervals and for some time he has been entirely free from them. This case is too recent to prognosticate, but at some future time we will give a further report on it.

DR. POLLOCK: Case 7, Mr. J. D., aged 51, presented himself on January 6, 1923, with a history of hoarseness of six months' duration, gradually increasing. We first saw him when the voice was almost gone. Examination showed an irregular growth on the left vocal cord anteriorly. Biopsy by suspension laryngoscopy on January 8, 1923, showed squamous cell carcinoma. On January 17th, under synergistic analgesia, the larynx was exposed and the left thyroid cartilage removed. Through this opening the growth was dissected widely and the wound closed. There was no tracheotomy, and he made an uneventful recovery. He went to work but presented himself monthly for examination. On July 31st a small fistula was present in the skin just over the external incision, extending into the larynx, through which mucus was discharged. There was no evidence of recurrence. On April 15, 1924, the fistula

was closed in the usual manner and up to the present time has remained so. Monthly examination shows no recurrence. He has been working the entire time and, as you notice, his voice, although a trifle husky, is good. It is now 27 months since the operation and he has remained free from recurrence.

Case 8.—Mr. L. E., aged 54, presented himself on January 22, 1924, with a history of hoarseness of eleven months' duration and some pain in the throat. He had been treated by a laryngologist for six months. Examination showed an irregular growth on the left cord. Biopsy showed a squamous cell carcinoma. On January 28, 1924, under synergistic anesthesia, a window resection of the left thyroid cartilage was done. The upper rings of the trachea above the tracheotomy opening were not removed. He made an uneventful recovery, and was then given a course of deep X-ray therapy and continued to give satisfactory reports until December 11th, when he complained of difficulty in swallowing. A stomach tube passed on several occasions revealed constriction just at the upper opening. On January 25th, after continued difficulty in swallowing, esophagoscopy was done, showing a constriction some two inches from the epiglottis. Edema was present but no signs of malignancy. From January 25th until February 17th the patient's condition remained about the same, and a bougie was passed on several occasions to relieve constriction. On February 17th the patient returned, complaining of severe pain and loss of weight, and it was impossible to pass a bougie. A duodenal tube was passed, and the patient was fed by this method for some time. At that time the edge of the tracheotomy opening gave the appearance of a recurrence of the carcinoma, and a biopsy made on March 10th revealed a recurrence. On March 23rd the entire trachea was cut away with the radio knife, wide of the apparently affected area. At this time an esophagoscopy was done, and a portion of the esophagus was obtained by the biting forceps, which microscopically proved to be nonmalignant. The patient's general health is fair, although we are still not sure that there will be no recurrence.

DISCUSSION.

DR. JOSEPH C. BECK said that fifteen or more years ago they did a few laryngectomies, with fatalities immediately or soon

after the operation. Most of the patients had lung or mediastinal complications and succumbed within a week or ten days after the operation. They then continued to do the laryngotomies, but no matter what method was used most of the patients either showed recurrence or succumbed to metastases. Since Dr. MacKenty presented his method of laryngectomy and they added to that the synergistic anesthesia, they have done practically nothing but laryngectomy. He believed the outlook for carcinoma of the larynx, when recognized early, should be encouraging, particularly to the younger men who are trained well in surgery. The operation of laryngectomy is not difficult if it is done under local or synergistic anesthesia and the outlook is not bad. With the use of the MacKenty instrument for speech, he was sure there was great hope in this field.

Dr. J. HOLINGER asked if there had been any symptoms of pain up in the side of the head in any of these cases.

Dr. BECK said that there had been no symptoms of this kind, that they were all intrinsic cases of laryngeal carcinoma, and the pain toward the head is present when it becomes extrinsic.

Cerebrospinal Rhinorrhea.

Dr. OTTO J. STEIN presented a patient who had suffered from cerebrospinal rhinorrhea, with disappearance of the symptoms under treatment.

A Polish woman, aged 45, married, applied for relief of a nasal discharge coming from her left nostril. Two years ago she presented herself for treatment on account of some nasal obstruction and anosmia.

Examination showed a healthy looking woman of average weight. There was impaired hearing, accompanied by some tinnitus. Both drum membranes were practically normal; the tubes were open; air conduction was somewhat reduced. The Schwabach was somewhat reduced, the Weber was not lateralized, the Rinne was positive. The upper tone limit was lowered, the lower tone limit elevated. Whisper test for both ears was reduced about 20 per cent. Both middle turbinals were hypertrophied, with polypoid changes. The polypoid middle turbinals were operated with a snare. Great improve-

ment followed the operation; free breathing through the nose was established and the sense of smell was restored.

She stated that one year later a watery discharge appeared from the left nostril following a head "cold." At the time there were no accompanying symptoms, such as itching of the nose, sneezing, headache, vertigo or lacrimation. She visited several physicians and was under their treatment, but the discharge never ceased, except for a period of a few days, when there was a decrease in the amount of fluid. This was while taking some "pills" which were prescribed. A renewal of this prescription, given for the menopause, failed to relieve the nasal discharge. Six months after onset of the discharge she consulted Dr. Stein. Examination showed a well preserved, healthy looking woman, not very nervous, holding a much soaked handkerchief to her nose, from which was dropping a clear, colorless fluid. She stated that the fluid never ceased, but varied in rapidity, especially on changing position of her head. With head forward or far backward the flow increased. It always came from the left nostril, soaked the pillows and towels at night and flowed into the throat, where it was swallowed. Many handkerchiefs were used each day, but after drying remained soft and pliable. There was an accompanying slight headache, which was not influenced by the amount of the flow from the nose. The blood pressure was 110 systolic. The eyes showed a mixed astigmatism. X-ray examination of the head showed all sinuses clear. A test tube full of the dripping fluid was collected in a few minutes' time and sent to the laboratory, where examination showed a water clear solution with no sediment. The Wassermann reaction was negative. There were no cells, no mucus, but traces of albumin and dextrose by both the Fehling and Benedict tests on two occasions.

Atropin internally had no effect. Alcohol injection of the left Meckel's ganglion was performed with no result. Benzyl benzonate solution, 20 drops three times a day, was administered, and after two weeks the discharge had almost ceased, after three weeks it entirely disappeared, and there had been no recurrence in about five months. The patient had a feeling of wellbeing and no increase in the headaches.

This disorder is comparatively rare. It had been his fortune to observe four cases in the past thirty years. Two were of

traumatic origin and both terminated fatally. In the other two the cause was unknown. Its etiology in the nontraumatic cases is obscure. It is possible that meningitis, hydrocephalus and brain tumor play a rôle in its causation. The discharge may persist for months or years without complete cessation. The only other symptoms associated with the disorder are headaches and perhaps some mental symptoms. With a remission of the discharge these symptoms are supposed to increase in severity and, conversely, they should improve or disappear with the re-establishment of drainage. This was not so in the case just reported. Headache was never a great factor, and there was no headache present after entire cessation of the discharge for the past five months. The rate of discharge varies from a slow drop to a very rapid dripping. A two ounce bottle has been filled in an hour's time. One quart has been discharged in twenty-four hours. A dozen or more handkerchiefs may be soaked during the day, but they are never stiff or harsh after drying like one containing mucus or other discharge. Sufficient drainage may be swallowed to cause nausea and diarrhea. If there is a perforated drum membrane the discharge may escape from the ear. The fluid has no viscosity, a faint alkaline reaction, a specific gravity of about 1.005; it contains about 1 per cent of solids, consisting of globulin, albumose, dextrose, potassium salts, phosphates and urea and a few lymphocytes. One must differentiate this disorder from other forms of rhinal hydrorrhea. In the latter there is usually itching of the nose, sneezing, the discharge ceases while asleep, the handkerchiefs become stiff on drying, the fluid has a viscosity, appears thick and sticky, opalescent, precipitates mucus and mucin, and does not reduce by any of the sugar tests.

The treatment that has been suggested and employed includes lumbar puncture, roentgen therapy, and the internal administration of thyroid extract, atropin or benzyl benzonate.

DISCUSSION.

DR. JOSEPH C. BECK said that he saw a female patient who was injured in an automobile accident. Following this she developed a cerebrospinal rhinorrhea, but was perfectly comfortable when her head was held back. If she leaned forward

the discharge occurred. He had seen two cases dissected by Chiari which showed deficiency at the cribriform plate, in which a fistula occurred and never closed.

DR. HARRY L. POLLOCK recalled the fact that he presented a very similar case before the society in 1924. The patient was a young lady who was perfectly healthy. She went to a party and danced a good deal and the next morning noticed that her nose was running. This was continuous except when the head was held in the erect position. She consulted him because of a cough at night, which was produced by the fluid running down her throat when she was lying down. Careful examination showed the fluid forming at the cribriform plate. He looked up the literature on the subject and instituted treatment with potassium iodid. After a few months the discharge ceased, and there had been no recurrence so far as he knew. The examination showed the discharge to be cerebrospinal fluid of low specific gravity.

DR. STEIN, in closing, said he thought everyone agreed that these cases were usually beyond relief. There may be a cessation of the discharge, but it almost invariably recurs. Posture has had a great deal to do with the discharge in all the cases, and in his patient the discharge was worse with the head bent forward or back.

Presentation of Specimen from a Case of Ludwig's Angina.

BY ALFRED LEWY, M. D.

The patient from whom this specimen was taken was a man, aged 21, six feet four inches tall, and well proportioned. He had a history of hemorrhagic effusion into the knee joint following a slight injury, and various other bleedings, including hematuria. Two of his mother's brothers and two of her uncles were known as bleeders, and his brother also has a history of severe bleeding from slight injury. March 18th he was seen by a physician, who diagnosed follicular tonsillitis. March 20th, at noon, he was again seen by his physician, who then thought quinsy was developing. Dr. Lewy saw him that evening in consultation. At that time his tongue was forced up against the roof of the mouth by a hemorrhagic effusion into

the floor of the mouth, which extended down the right side of the neck. Breathing was becoming increasingly difficult, the patient holding his head forward in order to breathe. He was transferred to the hospital, where he was given three ampules of fibrogen subcutaneously and calcium chlorid intravenously. The blood clotting time was seventeen minutes before this procedure and remained about the same afterward. Tracheotomy had to be done with the patient sitting up and with his head projecting forward. In this Dr. Lewy had the advice and assistance of Dr. C. E. Kahlke. The trachea was reached at a depth of about two inches and was opened just below the cricoid. The trachea was dislodged to the left, so that the intermuscular fascial planes could not be followed, and because of the position of the head the thyroid isthmus had to be retracted downward out of the field. The longest tracheal tube available at the time was barely long enough to reach when pressed inward by the thumb, so a catheter was passed through the tube, which carried him through the night. The next morning an extra long tracheal tube was procured which gave him fair breathing. He was transfused with whole blood by Dr. Bernard Portis. The pulse, which had been barely perceptible, did not improve much and the patient died that afternoon.

The specimen showed an enormous infiltration of hemorrhagic fluid, filling the entire floor of the mouth; both pyriform sinuses were filled flush with the epiglottis, which was folded on itself so as to close off the glottis; within the larynx itself was seen the same submucous hemorrhagic infiltration and section of the tongue showed that it extended into the tissues of the tongue itself.

Although there was oozing all night from the tracheal wound, and there was some blood found in the bronchi, Dr. Lewy did not think, from the appearance of the viscera after death, that death was due directly to acute anemia or to suffocation, as the patient's breathing was quite free up to the time of death. He was inclined to the opinion that death was due to pressure on the vagus or to toxemia. The white blood cell count was 25,000. He had no report on the type of bacterial infection.

Presentation of X-Ray Film of Submucous Ivory Implant for Atrophic Rhinitis.

BY HARRY L. POLLOCK, M. D.

A stereogram of a case of atrophic rhinitis in which a large piece of ivory was implanted in the septum submucously and the X-ray showed plainly the implant. He stated that for some time past they had been using ivory implants for the correction of external deformities with very good results, so he decided to try it in atrophic rhinitis. The patient showed marked improvement after the implant, and at this time, three weeks after the operation, the nose was nicely healed.

A Hemiplegia Following Antrum Irrigation.

BY JOHN A. CAVANAUGH, M. D.

A patient, aged 35, consulted me on February 10, 1925. She had suffered with a cold in the head two weeks before, followed by a pain over the right side of the face and back of the head, with nasal discharge of pus from right side. Dr. Otto Stein had washed out the right maxillary sinus two days before, with positive findings. The patient came to me to have the sinus washed again, as Dr. Stein had left the city.

A small pledget of cotton with three drops of cocain solution, 7 per cent, without adrenalin chlorid, was placed in the middle meatus over the hiatus semilunaris area and left for about ten minutes. I removed it and tested the area, and she complained of it hurting, so I introduced a cotton applicator, with two drops of cocain solution, 7 per cent, and let it remain again for about ten minutes. The patient was very nervous, yet controlled herself quite well. I then introduced a Pierce cannula into the lower part of the hiatus, and it dropped into the cavity; not a drop of blood appeared; the head and body were bent forward; the adaptor of the continuous rubber bulb syringe, 4 ounce size, was connected with the cannula, and an iodine solution, 1 to 12 in sterile water, was used for irrigating. The nurse, squeezing the bulb, said it worked very hard, and the patient complained of pressure. I judge about 4 drams of the solution returned from the sinus with some small globules of mucopus, then stopped entirely. I removed the bulb and applied the adaptor on a 4-ounce rubber bag, squeezing

the bulb carefully myself, and the air with iodine solution came through without a complaint of the patient and with but little pressure. I then reapplied the tip of the rubber irrigating bulb, and as soon as the nurse started to force the solution into the sinus the patient cried out, "Oh, my eye," and with that threw her head and body back into the chair and her legs and feet outward. Thinking she was in a faint, I put her head forward and low; there was no rigidity, no apparent respiratory involvement, no cyanosis; a pallor like that of a fainting person was present, the pulse was thready and weak. Aromatic spirits of ammonia were held at the nostrils, she seemed to take a deep breath, and the pulse improved. The pupils were not dilated and responded to light. There was no deviation and no nystagmus. She perspired very profusely. We placed her on a couch, and Dr. Orcutt tried to administer spiritus frumenti, but was unable to get it into her throat, as her jaws were closed. She was then given 1/100 of a grain of nitroglycerin hypodermatically. While on the couch her eyes converged mostly to the right, but would occasionally roll to the other directions, and she seemed to improve and respond to voices. About ten minutes after the nitroglycerin had been administered she vomited a large quantity of undigested food, after which she again appeared to improve. She was moved from the office in an ambulance after some time, still unconscious. At the time I thought it was a case of hysteria.

DISCUSSION.

DR. MAURICE LEWISON said that this patient was first seen by him on February 10th, at 5 p. m., with a history that while her antrum of Highmore was being irrigated a few hours before, she suddenly went into collapse and had been in that state ever since. She was completely unconscious, ghastly pale, and her expression was cadaveric. Vomiting was frequent. The eyes were open and staring, there was a conjugate deviation to the right, the right pupil was slightly larger than the left, and both pupils reacted to light. There was a complete quadriplegia, and none of her members responded to pin pricks. The reflexes on the right side were exaggerated, and the Babinsky was present. On the left side the reflexes were decreased, and at times the Babinsky was also positive. A care-

ful general physical examination revealed no abnormalities in throat, heart, lungs, abdomen or genitals. The pulse was 110, temperature 97.6° F., and respiration 16. The Kernigs were positive on both sides. The condition remained unchanged, and the following evening, after consultation with Dr. Olkon and Dr. Bassoe, neurologists, a spinal puncture was performed. The fluid came clear and at a normal rate. The patient's condition then became worse, and muscular twitchings developed in the left upper and lower extremities. These gradually increased, and she soon developed general convulsive seizures, involving the left face and upper and lower extremities. Ocular deviation to the right was more marked during that period, and retinal examination was negative. Dr. J. C. Beck saw the patient at this time and examined the antrum, which he found inflamed, and by suction drained a small amount of bloody fluid. At this time Dr. Frederick Tice and Dr. D. M. Trace also saw the patient, and they all agreed that she had developed a severe pathologic process in the brain. A hypodermic of morphin was administered, after which the convulsions gradually subsided, and a few hours later had disappeared and the general condition of the patient seemed slightly improved. She remained in about the same condition, being completely unconscious for about two weeks. During this time the temperature ranged between 100 and 101, pulse about 120, and respirations from 24 to 30. The motion in the right side of the body gradually improved, but the Babinsky remained positive on that side. The left arm and leg were completely paralyzed until about four weeks after the onset, at which time the motor condition gradually began to improve. Consciousness began to reappear two weeks after the onset, and gradually improved until she became absolutely clear mentally. The patient was still very weak, but the motor power of the right side was perfect; that of the left side was still impaired but gradually improving. During the course of the disease she developed a slight cystitis, due to catheterization and involuntaries, and also a cellulitis of the thigh, due to many hypodermic injections of camphorated oil.

Dr. Lewison believed a careful review of the symptoms, physical signs and circumstances under which this case occurred would at once suggest that a sudden vascular disturb-

ance in the brain occurred. Direct traumatism to the brain could be excluded by the fact that irrigation was under low pressure and that the anatomic conditions made such a possibility most improbable. The sudden vascular conditions to be considered were hemorrhage, embolism and thrombosis. Hemorrhage could be fairly well ruled out by the age of the patient and the absence of other evidences of vascular disease. Thrombosis could also be ruled out for the same reasons, and by the fact that the Wassermann examination of the blood and spinal fluid proved negative. Embolism was therefore the most likely pathology in this case. Septic emboli detached from the infected antrum, in order to reach the brain, would have to go through the pulmonary circulation and cause pulmonary infarction, a condition which is practically always fatal. Furthermore, very careful physical examination failed to reveal any endocarditis or other source of infection.

Air emboli could pass through the lung and lodge in the cerebral circulation, giving rise to pathologic changes and clinical symptoms such as were present in this case. This explanation was in keeping with most scientific researches on this subject and had recently been emphasized by the work of Grove of Milwaukee, who had made a study of a large number of accidents occurring during the washing of the antrum. He believed that this was an unusual case of multiple air emboli in the brain, due to the entrance of air in some of the veins of the maxillary sinus.

DR. JOSEPH C. BECK said that he saw the patient about 1 a. m. and found her in a supine position, jerking about two to the minute. The right arm was bent, the right eye showed conjugate deviation and there was nystagmus to the right. He introduced a trocar through the inferior meatus, and by means of Bier's pump withdrew a quantity of slightly blood tinged fluid. He could not see that the antrum was inflamed, and the patient did not improve following this procedure. The following day he saw her again in company with Dr. Cavanaugh. She then seemed to have improved somewhat. There was no change in the fundi, the pupils seemed to react quite well. He saw her two or three days later, and at that time she seemed to respond by grunts when spoken to. In his opinion, there was not enough air pressure used to account for the con-

dition. The blood picture showed 35,000 leucocytes, which he considered evidence of an infection, probably thrombotic in character.

DR. OTTO STEIN said that when he saw the patient he passed a Pierce cannula without any difficulty whatever. The woman was hypersensitive, and he examined her under thorough cocaineization of the nostrils, using the endoscope, and thought he recognized pus coming from the sphenoid, but irrigation of the sphenoid revealed nothing. He then introduced the cannula into the antrum, using a rubber bulb syringe, and being very careful not to introduce any air. The fluid entered the antrum and washed out an enormous amount of pus, following which the patient went home feeling relieved.

DR. MICHAEL GOLDENBURG (by invitation) said that he had a similar case in a female patient of about 35 or 40, who consulted him because of difficulty with her eyes. Examination showed some blurring of the optic nervehead on one side. Transillumination of the sinus disclosed a shadow in the region of the antrum. A trocar was passed, a washing made and just a small amount of air under pressure used. The woman dropped her head on her chest, there was a little quivering of the arms and legs. They thought it was an epileptic attack and gave some aromatic spirits of ammonia, but the patient did not respond. She was placed on the floor and artificial respiration was continued for an hour, but she never recovered. Their impression was that death had occurred because of the cocaine. The case was made a coroner's case and postmortem revealed air embolism in the heart, in the coronary, cerebral and mesenteric arteries.

They thought the probabilities were that some granulation tissue was present in the antrum and, by forcing in the air into the antrum, the thin walled vessels were broken and the air distributed in that way. The patient was probably dead before they resorted to artificial respiration.

DR. ALFRED LEWY asked if Dr. Cavanaugh observed any edema of the eyelids immediately after washing out the antrum.

DR. JAMES E. LEBENSOHN said that the amount of air used was of immense importance. Grove, in his paper given before the society two years ago, cited twenty deaths following the

use of air in the antrum. In many of the cases reported, compressed air was used under high pressure. The immense amount of air and the speed with which it entered into the blood stream caused the immediate fatality. Where less air was used, alarming sequences followed but the patient generally escaped with his life. Dr. Lebensohn knew of a case where even the air in the tube connecting a Lichtwitz needle with a pint piston syringe was sufficient to cause temporary hemiplegia, from which the patient completely recovered after a few hours. As to the pathology, Grove thought the needle separated the mucous membrane from the underlying bone, and that air was thus forced into the submucous venous plexus. Where symptoms occur after irrigation of the antrum, and even a little air is used, this air must be considered as the most probable factor.

DR. NORVAL H. PIERCE said that he saw a patient with a sinus infection and pus from the ethmoid, who had had a pledget of cotton soaked in cocain placed in the middle meatus. She waited in an adjoining room until the cocain had time to penetrate, and when she was called into the consultation room and attempted to walk she fell forward, apparently unconscious. Her husband was called and he took her away after some little time, still semiconscious. She remained in this condition for two days, and then gradually improved and recovered completely. In this case the antrum was not washed out, there was no instrumentation in any way, except that the pledget of cotton was placed in the middle meatus. She was a highly hysterical woman, with many peculiar mental quirks. In his opinion, there are cases of grave functional disorder which might simulate such a picture, even as that which occurred in this case of Dr. Cavanaugh's. Before concluding that the injection of just a small amount of air under the mucous membrane could produce so many pathologic conditions throughout the body, with paralysis of the leg and arm on one side and weakness on the other, without any respiratory or cardiac symptoms that would seem to be logically connected with an embolus, other possibilities should be considered.

DR. GEORGE W. BOOT said that as there are so many fatalities from washing out the antrum, he thought it was time to change the method. In the case reported, the patient was ex-

amined by two or three neurologists, who should be able to tell whether it was hysteria or not. It is so easy to open in the middle meatus with a rasp and wait a day or two before washing out that he considered this much the better plan.

Dr. CAVANAUGH (in closing) said that there was no disturbance or edema in any part of the eye at any time. In practically all of the cases in the literature there was a great deal of trauma, a needle puncture having been made, followed by bleeding. In this case the instrument used was a Pierce cannula, which dropped into the cavity. There was no trauma, no bleeding. The patient made no complaint until they started the water irrigation. Dr. Cavanaugh did not believe the condition was due to air.

Lung Abscess Following Safety-Pin in Esophagus

By JOHN A. CAVANAUGH, M. D.

A man, 22 years of age, Polish descent, employed at the steel mills, was sent to me on July 26, 1924, through the courtesy of Dr. Harry Parker of Gary, Indiana.

He stated that he awoke about 4 a. m. on July 26, 1924, with a pain in his throat, and thought that while he was asleep a big bug had crawled into his mouth. He tried to wash it down with water, but the water came back. His mother then prepared ground crackers and hot coffee, which also came back. After trying several other home remedies without relief, they consulted a doctor who had a private hospital in Gary. The doctor made an X-ray examination and discovered the safety pin lodged in the esophagus. The patient was advised to take an anesthetic and have the foreign body pushed into the stomach, and removed by an operation. The patient went home and talked the situation over with his father and mother, and decided to have this done. He returned to the doctor about 8 a. m., was put to sleep with ether, and the doctor tried to push the pin into the stomach. After over an hour of this manipulation another X-ray examination revealed the pin in the same location. The boy was then put back to bed, and when he had recovered from the effects of the anesthetic he was told that they had failed to get the pin, but would put him to sleep again that afternoon and remove it. He left

this hospital and went to the Mercy Hospital at Gary, where another X-ray examination was made, showing the safety pin. Dr. Harry Parker of Gary was called, and he advised them to get in touch with me. The patient was taken into St. Luke's Hospital, Chicago, and I saw him about 9 o'clock that same evening. He was sitting up in bed, had an anxious expression and was perspiring freely. He had a slight hacking cough, was spitting sputum streaked with blood, and it was difficult for him to talk, as it pained him more and increased the frequency of the cough. When spoken to he would respond in a whispered voice. He could not lie down, as it hurt him too much, and also caused more severe coughing. His temperature was 100.6° F., pulse 112 and respiration 30. X-ray examination showed a large safety pin opened with its point and shield uppermost, located from about the center of the fourth cervical to the upper edge of the seventh cervical vertebra. (Plate I.) He was given $\frac{1}{4}$ grain of morphin sulphate and 1/150 grain of atropin. Examination was made in the sitting position, as he was unable to lie down, and showed that the lips and gums were somewhat swollen and the teeth were covered with sordes. There was a small abrasion back of the incisor teeth on the hard palate, probably the result of a mouth gag. The tongue was somewhat enlarged and coated. The pharynx was bluish, swollen and coated with a blood stained mucus. Depressing the tongue showed the epiglottis and aryepiglottic folds also swollen and bluish.

With a Jackson direct laryngoscope, I examined the larynx. The vocal cords showed considerable redness, and the arytenoids were swollen, bluish and covered with a bloody mucus. The introitus into the esophagus had lost its normal appearance. No anesthetic was used. Having to talk to the patient through an interpreter made the work especially difficult. I introduced a Jackson 10 mm. esophagoscope and experienced no trouble in entering the esophagus. The esophageal mucous membrane was edematous, and the instrument looked as though it was in the center of an angiomatous field. I could not locate the foreign body, and when the tube was introduced between 19 and 20 cm. from the teeth line the patient became unruly and the tube had to be removed. I then decided to use the fluoroscope. As the patient could not lie on his back,

I had to have him lie on his side, which was exceedingly awkward. I used the Jackson esophageal speculum (Plate II), and when my assistant told me that the end of the instrument was at the shield end of the pin I carefully pushed aside the swollen tissue with a heavy alligator forceps, and finally spied the straight part of the pin between the shield and the spring. I grasped it and gently pushed the pin downward a little, when the point came into view. I was then able to get the end of the speculum under the point and then gradually withdrew the pin upon the speculum and removed it in this position. (Plate III.) The patient was then put to bed and could now lie down. Rectal feeding was ordered. The following day the temperature ranged from 99.6 to 100.4 degrees F.

On July 28th the temperature ranged from 98 to 99.6 degrees F., and the patient refused rectal feeding. He was then given bismuth subnitrate and calomel, as suggested by Dr. Jackson. Chest examination was negative.

On August 2nd he was taken home, as he refused all medicine and the nurses could not control him. Chest examinations were again made and found negative.

I heard nothing more from him until he returned to St. Luke's Hospital, three months later, under Dr. Arthur R. Elliott's service, complaining of cough, fever in the evenings, night sweats and weakness. When he was admitted into the hospital his temperature was 99° F., pulse 108 and respirations 28; the blood pressure, systolic 110, and diastolic 70. The patient stated that three weeks before coming to the hospital there was a sudden gush of very foul smelling matter up into his mouth. None had appeared since, but he had coughed up daily a greenish pus. Examination showed that the chest was symmetric; the excursion was good on the left side. The right side lagged and was shallow. On percussion there were no areas of dullness anteriorly. An area about two inches square was noted at the base of the right lung just to the right of the spine. Upon auscultation there was an absence of breath sounds over the area of dullness, an area of hyperresonance and loud sonorous râles up to the lower angle of the scapula. Blood examination revealed a leucocyte count of 14,600; hemoglobin 75 per cent.

The X-ray examination (Plate IV) showed a circumscribed mass involving the lower half of the right chest. The mass extended outward to the midclavicular and upward to the third rib anteriorly. A diagnosis of lung abscess was made.

Treatment by gravity was tried. An X-ray examination (Plate V), about three weeks later, showed a mass in the same position, but the density was much less and a very thick capsule was seen. As this procedure wrought no perceptible improvement, the patient was transferred to the surgical side, under the care of Dr. Albert E. Halstead, on December 16, 1924. Daily sputum examinations for tubercle bacilli were negative.

On December 22, 1924, Dr. Halstead resected two inches from the ninth and tenth ribs on the right side, close to the spine, exposing the posterior mediastinum. There was no adhesion between the visceral and the parietal pleura, and the abscess cavity could not definitely be located. Packing was introduced to cause an adhesion of the parietal and visceral pleura. A ring was placed at one point to determine the relation of the abscess to the area packed, and X-ray examination (Plate VI) showed the ring at the lower and external part of the dense mass. A week after the resection of the ribs an incision was made at the ring site into the supposed abscess; no pus was found, but necrotic lung tissue of a very foul odor. Dr. Halstead removed this necrotic tissue and then packed with tincture of benzoin on gauze. Following this the patient gradually improved. An X-ray examination (Plate VII), on January 20, 1925, showed the dense area no longer sharply defined and considerably cleared. The patient left St. Luke's Hospital February 10, 1925, and is now back at his work in the steel mills.

DISCUSSION.

DR. EDWIN MCGINNIS thought that the X-ray examination made foreign bodies look very easy of removal. He congratulated Dr. Cavanaugh on getting the pin out without any additional trauma and upon the happy result of the case. He asked if any bacterial examination of the expectorated material was made.

DR. CAVANAUGH replied that a fusiform and spirochete was found.

DR. MCGINNIS stated that he had recently reviewed the findings in similar cases, and there was usually great difficulty in securing a satisfactory external drainage. In one case the surgeon made two or three attempts to get the tube into the abscess cavity, and when the tube was in place very little pus drained out. In view of the work that had been done, he thought the best way to drain such abscesses was through the trachea, by means of a bronchoscope.

The intravenous injection of neoarsphenamin has also been of benefit in these cases. He had a similar case under treatment, except that the foreign body was a blood clot in the bronchus.

DR. GEORGE W. BOOT thought there was nothing more difficult than removing open safety pins from the esophagus. He has had four bronchoscopic deaths, two of which were due to safety pins. In an infant seen recently the pin was accidentally touched with the tube used for removing the saliva and dislodged. The procedure was stopped and the doctor said they would do nothing until the next day. By the following day the child had passed the safety pin with the stool. The ring end is sure to pass first and does not produce much trauma.

The youngest patient he ever had to remove a safety pin from was an eleven-day-old infant, who swallowed an open safety pin. X-ray examination showed the pin open to a right angle. Dr. Boot shoved the esophagoscope down over the pin and removed it by drawing the pin into the tube and straightening it.

Dr. Boot presented roentgenograms of several other infants who had swallowed safety pins, and one who had swallowed a cork.

He said Dr. Cavanaugh did not mention the relation between the lung abscess and the safety pin in the esophagus. Dr. Boot did not believe there was any relation, except that the young man had received two anesthetics before Dr. Cavanaugh saw him, and that the abscess was due to inhalation while under too deep an anesthetic. He did not think the safety pin itself had anything to do with the occurrence of the abscess.

DR. FRANK NOVAK, JR., asked what was the relation of the site of the safety pin to the site of the abscess.

DR. CAVANAUGH (in closing) said, in reply to Dr. McGinnis, that probably a bronchoscopic examination immediately would have been a good thing, but as the case turned out he doubted if it would have resulted in any benefit. The patient was given three doses of neoarsphenamin at different times following the operation.

Replying to Dr. Novak, he said that so far as he could see, there was no relation between the location of the pin and the formation of the abscess. There was nothing in the roentgenogram which showed any connection.

Dr. Cavanaugh agreed with Dr. Boot that the abscess was probably due to the inhalation of some foreign material during the ether anesthesia.

Insulin in Diabetes With Mastoiditis.*

BY T. C. GALLOWAY, M. D.

DISCUSSION.

DR. CAVANAUGH and DR. PIERCE congratulated Dr. Galloway on his presentation.

DR. GEORGE W. BOOT thought that the infection by streptococcus hemolyticus was very important, and considers this one of the causes of diabetes. In two cases of scarlet fever which he had seen the patients developed diabetes as the result of streptococcus hemolyticus infection.

*See page 1184.

AMERICAN OTOLOGICAL SOCIETY.

The Present Status of the Operation of Removal of the Stapes for Vertigo.

BY EUGENE A. CROCKETT, M. D.,

BOSTON.

Most operations for relief of vertigo are very severe and present reasons for surgical hesitation. The removal of the stapes, from its niche in the oval window, is not dangerous, although it requires nice technic and a very careful selection of cases. Prolonged and distressing vertigo is often due to inelasticity of the apparatus of the internal ear, caused by adhesions in the middle ear. These cases are capable of giving the most satisfactory operative results. The following cases may be cited:

Mrs. S., 66 years, excessive vertigo, deafness and tinnitus in left ear, left membrana tympani thickened and retracted. Left stapes removed, with complete relief from vertigo since operation.

Case 2.—Mrs. H., 58, deaf, right ear, 12 years, worse past 18 months; for five months has had vertigo, nausea, vomiting; staggers to right in walking. The caloric reaction was slow and shortened in right ear; hearing decreased. Stapes operation, since which time no vertigo.

Care must be taken to exclude cases of otosclerosis or of eighth nerve tumor in this group, also cerebral syphilis, locomotor ataxia and multiple sclerosis.

The after results of this operation are excellent in well chosen cases, which are often the type most intractable to ordinary methods of treatment. These symptoms are so incapacitating to the patient that some simple method of relief is most desirable. In the hands of a trained aurist there should be 75 per cent of recoveries. The only complication noted is the danger of causing facial paralysis by accidental lesion of the seventh nerve.

Three Cases of Otitic Brain Abscess.

BY S. MACCUEEN SMITH, M. D.,

PHILADELPHIA.

The early stage of this disease is usually ushered in by well known symptoms, such as pain, nausea, projectile vomiting, with decrease or cessation of otorrhea, but some cases are atypical and misleading as to diagnosis. Some valuable diagnostic signs are: A change in disposition, from brightness to moroseness, or vice versa; a lowered, even subnormal, pulse rate, temperature and respiration. Yet these do not always occur. Some cases simulate sinus thrombosis and some meningitis.

The first case reported in this group was that of a prominent engineer, seen first January, 1923, with a history of intermittent discharge of the ear for nine years. If the otorrhea stopped he had intense vertigo and mental confusion. Removal of a polyp from the external canal decreased the discharge and the vertigo. The discharge, however, suggested a necrotic mastoid, and he was advised to have an operation. He agreed, but the operation was postponed for over a year. Following the operation the patient grew worse, and I was called in consultation. I suspected brain abscess. He improved sufficiently to be up and around, but he later suffered from diplopia and temporofrontal headache as well as Bell's palsy. A second mastoid operation relieved these symptoms for two months, when he again became ill with symptoms indicating intracranial pressure, but without localizing symptoms. For some time the patient alternated between periods of stupor and comparative mental clearness, but his condition became poorer, so that operation was postponed. Drainage of the ventricle gave relief from pressure, but the patient died shortly after. Autopsy showed cerebellopontile abscess of the left side.

Case 2. Temporosphenoidal Abscess.—Male, 43, admitted to hospital September 10, 1924. At 7 years old, 36 years ago, the patient had acute suppurative otitis media, right, which ruptured and drained for a year, then cleared up. Since then he often removed "wax" from the ear. Thirty years after the first attack he had influenza, with increase of aural discharge.

Four years later he suffered an attack of severe right sided head pains. No discharge, no dizziness. Eyes and reflexes normal. Twenty-four hours later he had chills, fever, rapid pulse, severe pain, then a profuse discharge occurred which relieved the pain. He returned to work. Ten days later he was injured on the head, which caused return of symptoms. These continued, off and on, for a month, with increasing severity, until signs of meningitis occurred and he was taken to the hospital, where a diagnosis of brain abscess was made. An abscess was found in the temporosphenoidal lobe and evacuated through the mastoid wound. He improved, but died of pneumonia two days later. He probably had had brain abscess for a considerable time. He might have been saved by earlier operation.

Case 3.—A child, 7, who had suffered acute otitis media at $4\frac{1}{2}$ years. At $5\frac{1}{2}$ she had influenza with multiple abscesses. Nine months later she developed severe pain in the head for several months, not referable to the ear until eight months later, when the right membrana tympani ruptured with relief of pain. She then suffered from facial palsy and bulging of right eye, with projectile vomiting, and was semiconscious when admitted to the hospital. A temporosphenoidal lobe abscess was evacuated through the mastoid wound. The child improved markedly for several weeks, when suddenly the symptoms returned, she had a violent convulsion and sudden death. This sudden fatal termination may have been due to embolus.

Twelve Cases of Cerebral and Cerebellar Abscess.

By H. P. CAHILL, M. D.,

BOSTON.

Twelve cases of cerebral or cerebellar abscess were operated on in all, six by myself personally and six by other members of the staff. About 60 per cent of the cases showed infection through the mastoid route. The cases were very carefully watched for signs of localization of the abscess, there being no attempt made at operation until the location of the pus was reasonably certain. Two of the cases operated on were very late ones and the patients were in extremis. The abscess had

existed for some time and had become well walled off. We ascribe the favorable results in all cases to the use of the Mosher drain, which is made in three sizes, and of which the medium size drain is most satisfactory. We use a trocar to locate the abscess, and the drain is immediately inserted. It is very valuable in acute cases of abscess without a wall, as it gives the opportunity for the wall to develop. On account of the cone shaped wire basket which forms the drain, the tendency is for the cerebral tissues to gradually expel the drain. The drain can be allowed to stay in for some weeks, and in most cases it has been forced out naturally, but we have yet to work out a technic by which the drain can be removed easily and avoid having the drain fixed in the tissues by granulations. We believe that there need be no hurry to make early diagnosis in these cases and that the important points are the localization of the pus and immediate placing of the drain. The results from the use of this drain are very satisfactory.

Some Remarks on Labyrinthine Surgery.

By E. B. DENCH, M. D.,

NEW YORK.

Cases are divided into those of, first, localized labyrinthitis without symptoms. Simple exposure of diseased area, and labyrinth left alone.

Second—Invasion of the labyrinth accompanied by symptoms. These call for drainage, either by the Hinsberg operation or by the Neumann operation, preferably by the former. Seven of these cases. All recovered.

Third—Cases in which the labyrinthine suppuration spreads to the meninges. These cases were all operated on by the Neumann method, with drainage of the subdural space. All died except one.

Accidental removal of the stapes, per se, does not necessarily call for operative interference. Packing off the region of the stapes by separate packing is ordinarily sufficient.

The presence of a dead labyrinth without symptoms does not necessarily call for labyrinthectomy at the time of a radi-

cal operation. The radical operation may be performed and the dead labyrinth left in situ without harm.

Extensive sequestra involving the labyrinth are not infrequently found in cases of chronic middle ear suppuration. Very few labyrinthine symptoms may be present at the time of operation, although the bony destruction may be extensive. These cases usually recover after operation.

Neuro-Otologic Examination in Seven Consecutive Verified Cases of Brain Tumor in the Posterior Fossa in Children.

BY D. E. S. WISHART, B. A., M. D.,

TORONTO.

Seven cases of verified brain tumor in the posterior fossa in children are reported. Four of these were definite intracerebellar tumors. Three were extracerebellar but in the midline. Three of the cases had precisely the same lesion. Complete histories and neuro-otologic details are given regarding each case. The data have been summarized in four tables to permit easy reference. Photographs of the brain tumors accompany the paper.

In view of the information gathered from this and from the previous series of cases, I would now submit that our evidence permits the following postulates:

1. Tumors of the frontal lobes do not interfere with cochlear or vestibular function.
2. Subtentorial tumors which are completely outside the brain stem and the cerebellum affect homolateral cochlear and vestibular function.
3. Subtentorial tumors which are in the midline and which do not involve cerebellar tissue do not affect cochlear function. They do interfere with vestibular function. The interference tends to be bilaterally symmetric, and the functions of both vertical canal reflex arcs are affected before those of either horizontal canal.
4. Unilateral intracerebellar tumors do not interfere with cochlear function. They do interfere with vestibular function. The function of the reflex arcs of the vertical canals will both be affected before those of the horizontal canals. The tumor

will be found on the side opposite to the vertical canals whose function is most interfered with.

The Present Status of Vestibular Tests in Intracranial Conditions.

By LEWIS FISHER, M. D.,

PHILADELPHIA.

The records of 103 suspected cases of brain lesion, seen at the University Hospital, Philadelphia, were examined and classified. The cases selected were verified by operation or autopsy, so that the diagnostic value of vestibular tests in such cases could be definitely estimated. The tabulation is as follows:

	Vestibular examination	
	Correct	Incorrect
Cerebellopontile angle tumors, 29 cases.....	27	2
Tumors of fourth ventricle, 3 cases.....	3	
Midline infratentorial tumors, 2 cases.....	2	
Cerebellar lesion, 28 cases (cerebellum ruled out by vestibular examination, 2 cases).....	21	5
Cerebral lesions, 28 cases (a) hemispheric lesions	*15	
Vestibular examination ruled out posterior fossa	10	
Diagnosis made by vestibular examination alone	3	
(b) Pituitary lesions, 13 cases.....	†10	‡1
Vestibular examination ruled out posterior fossa	2	

Otologists are particularly interested in cerebellopontile angle tumors, because they are first consulted in these cases. The phenomenon complex of a totally destroyed ear on one side, with nonresponsive vertical semicircular canals on the side opposite the lesion, and good responses from the horizontal canal on that side, was found to be reliable with few variations.

Vertigo and past pointing were reliable as to condition of posterior fossa contents. No definite phenomenon complex was

*Correct side. †Correct location. ‡Incorrect.

found for cerebral lesions, but if the patient had signs of brain lesion and was easily made sick by turning and douching, the case was cerebral. In conclusion, neurotologic tests conducted by otologists reached the same conclusions as neurologic examinations, and in some cases the otologic examinations were the only method of study by which diagnosis could be made.

The Importance of Analysis of the Complete Vestibular Response.

BY J. Q. HOLSOPPLE, M. D.,

BALTIMORE.

In order to correlate nystagmus time with ordinary responses involving change in position, the phenomena of the semicircular canal mechanism were studied in a group of males, undergraduate students, in good physical condition, between 17 and 25 years of age. The postrotation nystagmus times for all reactors, both clockwise and counterclockwise, were measured, the variations being from 19 to 34 seconds. A slow turning chair test, about a turn in 18 seconds, was given, and showed wide variations in estimation of a complete turn, in blindfolded students. A test on the campus was made with blindfolded students, headed for a tree at 262 feet distance. Wide deviations were noted. The results of these and repeated tests showed that all of the reactors had normal vestibular apparatus. Yet when deprived of visual aid they showed great tendency to deviation, either to right or to left, and that they were not aware of deviations. It would seem, therefore, that the vestibular stimulation must be of low intensity and of near threshold value. Thus the determination of threshold value for different individuals would seem of practical importance. We should have a measure of the minimal intensity of rotational stimulation required to provoke the vestibular response. These experiments should be extended to cover responses to rotation around other axes of the body, since other canals than the horizontal may affect orientation. The tests should be repeated until their normal variability is established and pathologic reactors should be compared with normal reactors in order to determine what extravestibular factors may affect the response.

In conclusion, nystagmus time is not the only nor the most striking of vestibular responses.

Dynamic Studies on the Cerebrospinal Fluid in the Differential Diagnosis of Lateral Sinus Thrombosis.

BY GEORGE L. TOBEY, JR., M. D., AND JAMES B. AYER, M. D.,

BOSTON.

This study is based upon two important statements: Increased intracranial pressure is normally propagated throughout the cerebrospinal fluid system and may be measured by a manometer employed in connection with lumbar puncture. Compression of the internal jugular vein causes increased intracranial pressure with immediate rise in spinal fluid pressure.

Absence of rise in fluid pressure (positive Queckenstedt sign) indicates spinal subarachnoid block (as in spinal cord tumor or cerebellar fossa tumor). Further extension of the use of this test is to detect evidence of block by lateral sinus thrombosis, partial or complete, thrombosis of the jugular bulb or internal jugular vein. The mechanism in venous obstruction differs from that by tumor, in that there is no elevation of pressure at its source, because of venous obliteration. The test for sinus thrombosis includes the examination of pressure effects on each jugular vein separately, and not with simultaneous compression of both veins, as in the diagnosis of cord tumor.

In a typical case of lateral sinus thrombosis there is a prompt and rapid rise in fluid pressure to twice or three times the initial reading, on the normal side, this pressure rise being maximal and equivalent to the pressure attained when both jugular veins are compressed. Pressure over the thrombosed lateral sinus causes either no rise, or a slow rise of only 10-20 mm. in the manometer. Partial obstruction gives less striking results, yet of value as corroborative evidence.

General septicemia is not a contraindication to the test.

The test is of special value in the presence of double mastoiditis developing symptoms of lateral sinus thrombosis.

Herpes Zoster Oticus: A Case with Involvement of the Fifth, Seventh, Eighth and Ninth Nerves with a Complete Vestibular Examination.

BY FRANK L. DENNIS, M. D.

Review of the pathologic studies of Herd and Campbell and Ramsey Hunt in herpes zoster, demonstrating the identity of the changes in the posterior ganglionic chain in herpes zoster with those of the anterior horns in acute anterior poliomyelitis. The experiments of Teague and Goodpasture, Lipschütz and Flexner suggest that the infectious agents of encephalitis, herpes simplex and herpes zoster are closely allied or are identical.

Infected foci of the upper respiratory tract as etiologic factors.

Description and distribution of the eruption in herpes zoster and herpes zoster oticus. In typical cases the geniculate ganglion of the seventh cranial nerve is involved, together with the motor branches, and often the eighth, ninth and tenth cranial nerves also.

Origin of all posterior spinal ganglia is the embryologic neural ridge; auditory ganglia also spring from the same source. The otic ganglion belongs to the sympathetic system and is not involved in herpes zoster.

Symptoms depend on the ganglia involved and are general and local. The auditory ganglia may escape or only one may be diseased. If Scarpa's ganglion is affected, vestibular apparatus will be unexcitable. If Corti's ganglion is involved there may be only tinnitus or hypoacusis, or complete deafness may result. Where there is pain and tenderness in the ear and mastoid with eruption on the drum membrane, otitis media is often suspected and paracentesis is done. Case report.

The Anatomy, Psychology, Diagnosis and Treatment of Congenital Malformation and Absence of the Ear.

BY JOSEPH C. BECK, M. D.,

CHICAGO.

1. Introductory remarks concerning the inadequacy of general and textbook literature on the subject.

2. The psychologic examinations of these patients as well as the parents.

3. The psychiatric examination with special reference to the inferiority complex.

4. The medical or pediatrician's examination with special reference to the general physical examination and management.

5. The otologic examination with special reference to the external deformity, the radiologic examination and the physiologic examination, particularly as to the internal ear.

6. Lantern illustration of eleven cases upon which this study is based.

7. The selection of five of these, (a) infant, (b) older child, (c) adult, with their radiograms, functional tests and summary remarks.

8. Conclusions.

A Clinical Study of Bone Conduction After the Method of Runge.

BY JESSE WRIGHT DOWNEY, JR.

Most otologists believe that there is such a thing as normal bone conduction—that is to say, that a vibrating tuning fork should be heard when its stem is pressed against the head for a length of time which does not vary greatly with one particular fork in any number of individuals. It has never been definitely determined whether bone conduction is simply the perception of those sound waves which pass through the sound conduction mechanism (Bezold) or whether there is a direct craniolabyrinthine transmission (Wittmaack and others).

In 1923, H. G. Runge published a study of bone conduction and described his "water filling" test, which is simply the timing of the duration of bone conduction with the stem of the fork over the mastoid before and after filling the ear with 1 cc. of water. In the normal ear or in the pathologic ear with a normal drum and ossicular chain, bone conduction can be doubled by the "water filling." The test has been repeated in a series of cases and the same results obtained. Runge, however, explained the reaction by claiming that increased bone

conduction was caused by a better conduction of sound waves through the fluid. This apparently has been disproved, and the theory is offered that the increase is occasioned by a change in position of intralabyrinthine structures brought about by the pressure exerted on the footplate of the stapes and the compensatory action at the round window. The Runge test also makes it possible to study artificially produced Weber and Schwabach experiments and suggests the possibility of estimating the bone conduction of one ear alone. The experiments offered are purely clinical experiments, but the observations offered suggest that there is a direct craniolabyrinthine transmission of sound vibrations, and that bone conduction is an entity which may be separated from physiologic air conduction, thus making the universal tests by bone conduction of more value in differential diagnosis.

Results in Radical Mastoid Operations as to Hearing.

BY PHILIP HAMMOND, M. D.,

BOSTON.

In transmission of sound waves to the auditory nerve there must be no impediment to vibration of the ossicular chain. The movement of the perilymph is necessary to nerve stimulation. After a radical mastoid too often granulations and discharge encroach upon the middle ear and become epidermatized. The organs of hearing are effectually sealed. If large formation of connective tissue occurs after skin graft, this same result happens. Another cause for damage of hearing is the persistence of raw, discharging surfaces in the middle ear. It has been found, from personal experience with patients, that where the sound perceptive apparatus is intact, the clearing away of debris will restore good hearing. I have had many cases with fortunate outcome and great improvement of hearing, and if care be taken in the radical mastoid operation to prevent impediments to stimulation of the nerve, these cases can get well and remain well, and can recover a degree of hearing which is highly satisfactory to the patients. Of course we also have our bad cases, which are very discouraging.

**Choked Discs in Association With Surgical Mastoid Disease Without
Apparent Intradural Involvement.**

By H. I. LILLIE, M. D.,

ROCHESTER, MINN.

The association of choked disc with surgical mastoiditis may be considered an important sign of intracranial or intradural infection. It has been noted, however, that choked disc may occur with surgical mastoiditis, without other evidences of extension.

Intracranial infection is not as serious as intradural infection, because the dura is highly resistant to infection, while the brain substance is not. It is wise, therefore, in cases of surgical mastoiditis with choked discs, that careful studies be made and that no attempt be made to interfere with the brain substance until evidence of intradural invasion is certain. While the incidence of choked disc in surgical mastoiditis without intradural extension is low, it is sufficiently frequent to be considered in the differential diagnosis.

Clinically four cases of the latter finding have been studied in detail. They recovered completely. There was no conclusive evidence of intradural extension, although it was not definitely excluded. Three patients had parasinus abscess, the fourth showed pachymeningitis, and a large cholesteatoma pressing on the sinus and cerebellum. In one patient intradural exploration was made without result. The mechanism of choked disc in such cases is a moot question. Unless we have strong evidence of intradural infection, exploration of the brain is not warranted. The fundi oculorum should be examined in all surgical mastoid cases.

Method of Skin Grafting for Radical Mastoid Cavity.

By HARRIS P. MOSHER, M. D.,

BOSTON.

I prefer to graft a radical mastoid cavity whenever possible, and for the past ten years I have used the paraffin basket mould for the following reasons: It is an accurate mould of the cavity; it carries the graft to all parts, over the facial ridge, to the floor of the canal and fits the irregular walls of

the middle ear; the mould can often be left in several days, which is of special advantage with children.

In cases where secondary operation is preferable, the mould can be put in to control the granulations until the operator is ready to graft. It is also possible to graft only the middle ear, and do it accurately, with the use of the mould. Another advantage is that the paraffin adheres to the graft sufficiently to smooth out the wrinkles and prevent rolling of the edges. It has also been found that paraffin has the property of protecting the epithelium and stimulating its growth.

The question as to whether paraffin would impede drainage and so indirectly bring about meningitis or infection of the lateral sinus has been answered in the negative. The heat of the body melts the paraffin enough to permit of drainage.

The method of making the paraffin gauze basket is as follows: Take a strip of gauze, 6 inches wide, 12 inches long, fold twice toward center, making a four-ply type $1\frac{1}{2}$ inch wide; dip in parowax, so as to obliterate the mesh of the gauze. Roll up, place in sterile container, ready for use. When wanted, unwind, softening if necessary in sterile warm water. Before using, the mastoid cavity is plugged with gauze soaked in adrenalin. A cone is formed of the paraffin tape, letting the cone edges overlap; the tip of the cone is filled with melted paraffin to take a mould of the middle ear and aditus. The gauze mesh must be thoroughly covered with paraffin or it will cause granulations. The mould is left in for hemostatic purposes while the graft is being cut. A second mould can be used to keep the cavity dry while the graft is being fitted on the first mould. The mould must be trimmed flush with the surface of the mastoid and not project, or it will interfere with primary healing. If a meatal flap is used it is tucked in place back of the mould, and the cavity of the mould is filled with ordinary gauze. The mastoid wound is closed, either wholly or a portion can be left open for insertion of a drain. The mould is left in place five to ten days. If the wound becomes puffy, red or discharging, the mould should be removed.

To remove the mould, take out gauze packing and collapse the basket sides. When the mould is taken out it is a great

satisfaction to see the well fitting graft left behind. The basket mould can easily be removed through the meatus.

In dressing the skin wound of the leg where the graft was taken, a number of strips of paraffin gauze are cut and moulded to form a plate. This is put over the skin wound and left in place four or five days. The epithelium grows rapidly under the paraffin plate and is removed painlessly, thus forming a most satisfactory dressing.

Some New Methods and Apparatus for Testing the Acuity of Hearing and Their Relation to the Tuning Fork and Whispered Voice Methods.

By HARVEY FLETCHER, M. D.

A method of reducing speech, whisper, watch tick, coin click, acoumeter and tuning fork tests to a common basis of comparison is given in the paper. The relation between hearing losses in sensation units and maximum distances for hearing is given with illustrative examples. It is shown that the normal distance for hearing and interpreting a voice of average intensity in a quiet place is about 1,300 feet instead of 40 feet as sometimes assumed.

It is shown that tuning fork data may be reduced to a common basis by the relation that the per cent hearing loss is equal to a constant of the fork multiplied by the difference in the hearing times for a person of normal hearing and for the person being tested. Typical results are given for three groups of tuning forks.

Three types of audiometers will be described and their operation demonstrated, namely, the phonograph types for making speech tests, the buzzer tone types for making a quick test of the general hearing level, and the tone range types for making audiograms to be used in diagnosis.

Results of hearing tests made on ten patients having hearing losses from 0 to 100 per cent, taken by the various methods discussed, are given. A comparison of these results leads to some interesting conclusions concerning the merits of some of the different kinds of hearing tests.

A simple method of calculating the hearing loss for speech from the audiogram, taken with the tone range audiometer, will be given.

The Potentialities of the Audiometer.

BY M. A. GOLDSTEIN, M. D.,

ST. LOUIS.

A study of the requirements for audiometers in regard to operation and utility includes: A tonal range of 16 d. v. to 24,000 d. v., or full limit of normal audition. Tone intensity: an increase in intensity to 200 dynes is required in testing the profoundly deaf. A bone conduction receiver is recommended to carry out bone conduction audition tests. Uniformity of records and methods of charting is one of the most important features of the work. The Western Electric Company's chart is practical and dependable. A plea is made urging the adoption of this scheme of charting. Tone intervals: it is urged that a seven step instrument be used, instead of one or two step, in order that more accurate information may be had as to tone islands and tone gaps.

Practical Application.—Conduction deafness: This is shown on the chart as a reduction in perception of the lower pitched tones in the hearing range. The high pitched tones will approximate normal. Perception deafness: This graph shows a diminished appreciation for the high pitched tonal range, the upper range being curtailed. After 50, in normal individuals, there is reduction to sensibility to the upper tone range. Otosclerosis: This shows reduction of sound perception for the entire tonal scale, with absence of tone perception for 32 d. v. and sometimes 64 d. v. of the lower tonal range, and also absence of perception for 8,192 d. v., and even 4,096 d. v. in the upper. Congenital deafness: We can test residual hearing in profoundly deaf individuals. The human voice lies between 300 d. v. and 3,000 d. v., and a child lacking perception for this is unfit for education by voice or instrument. Tone islands: Gaps for hearing certain portions of the scale, and most frequently above the upper limit of hearing for the human voice (4,096 d. v.), about the end of the middle whorl of the cochlea. This opens an interesting question of research.

**The Vestibular Tests as Employed by the Army.
(U. S.) Air Service.**

BY CHAS. A. PFEFFER,

CAPTAIN, M. C., U. S. ARMY.

This paper describes the technic of the Barany chair rotation test as well as caloric douche test of the vestibular apparatus. The qualifying limits of nystagmus rate, past pointing and falling, as prescribed by present army regulations, and the proposed new regulations, are discussed. Deviations from the commonly accepted normal reactions, in the pilot, are interpreted as a probable overcompensation on the part of the pilot for the falling and pointing reactions frequently observed—particularly in many constant stunt fliers.

Flying is not an ear problem, although the danger of flying with abnormal vestibular function is not to be overlooked.

Due importance in the maintenance of equilibrium to orientation is given to vision and body sense—i. e., tactile, muscle, joint, viscera, etc.

The internal ear is one factor in flying. The most important factors in the selection of the flier are the eye and the nervous system. The ear is of secondary importance in the case of the flier, the nervous system being the most important. Once the pilot has learned to fly, the ear is of even less importance.

Man is a native of terra firma. His love of travel and speed in his journeys are conspicuous. His efforts are ever toward greater velocity. A sculptor became immortal by putting wings upon the ankles of Mercury. Folklore and history set forth man's desire to fly with the birds in the clean, free domain of the air ocean, and in the vernacular common to localities we sense his irrepressible longing by the names bestowed upon excessively mobile units of his transportation. We had the "Flying Squadron" of fast naval vessels; the "Dixie Flyer," railroad train; the "Thomas Flyer," automobile, etc. Each of these hurried but did not fly. Man now lives in an era of actual flight, and wide attention has been given to examining and grading individuals to determine which could be successful in this new art. We, sadly, have learned some were not adept.

All through these years of the development of aviation it has been my sincere belief that of greater importance than normal nerve end organs is a cerebral ability to assimilate nerve information and coordinate and synchronize muscular activities suited to the positions or occasion. Irrespective of an individual's other accomplishments or wisdom, it is that ability which enables him to fly gracefully and guide his airplane consistently to smooth, soft landings.

The very extensive and painstaking work of the United States Air Service has shown that a great many student aviators who appear normal cannot become superlative pilots after 100 or more hours have been devoted to training them. It was to diagnose those latent faculties and accomplish their primary education that I evolved my orientator. In no sense should it be operated violently to see if the student can "stand" the gyrations of an airplane. Extreme gentleness and accuracy of operation is advantageous, and enables a diagnostician understanding it to estimate and improve a student's perception, cerebration, coordination, synchronism and delicacy of muscular activity.

Subnormal sensory fields may be more than compensated for by abnormal capabilities of other units active in the composite which, together, makes success.

By most rigorous trial at the primary flying school of the Air Service on a considerable number of men, the theory is found to be practical. It "works."

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